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(54) Title: IDENTIFICATION OF GENES INVOLVED IN ANGIOGENESIS, AND DEVELOPMENT OF AN ANGIOGENESIS DIAGNOSTIC CHIP TO IDENTIFY PATIENTS WITH IMPAIRED ANGIOGENESIS

(57) Abstract: The invention is directed to methods for angiotyping individual patients to predict the likelihood of whether a given individual will develop good vs. poor collaterals naturally. Accordingly, this can involve obtaining and providing a list of genes involved in collateral development. In particular, angiotyping individual patients can be used to predict the likelihood of whether a given individual will develop good vs. poor collaterals in response to specific angiogenesis therapy. From an array of genes that have been determined through experimental studies as being differentially expressed in tissues in which collaterals are developing in response to arterial occlusion, single nucleotide polymorphisms (SNPs), or other epigenetic changes, such as DNA methylation patterns, can be identified. SNPs and DNA methylation patterns are detected using microchips or similar technology assaying for all, or most, of the genes determined to play a role in collateral development. In addition, abnormally low or abnormally high differential expression of any combination of the candidate genes can be detected in such tissue as peripheral blood cells. The presence of a predisposition to develop poor vs. good collaterals is indicated by the presence of SNPs, and/or alterations in DNA methylation patterns, and/or difference in expression levels involving one or more of the genes.

IDENTIFICATION OF GENES INVOLVED IN ANGIOGENESIS, AND DEVELOPMENT OF AN ANGIOGENESIS DIAGNOSTIC CHIP TO IDENTIFY PATIENTS WITH IMPAIRED ANGIOGENESIS

This application claims priority to U.S. Provisional Application Serial No. 60/432,005, filed December 10, 2002, the contents of which are hereby incorporated by reference in their entirety.

Field of the Invention

The invention provides compositions and methods for the identification and isolation of genetic elements related to angiogenesis and to the creation and use of arrays containing isolated genetic elements.

Background of the Invention

Coronary artery disease and peripheral vascular disease are endemic in Western society. In these diseases the arteries that supply blood to the heart muscle or to the legs become narrowed by deposits of fatty, fibrotic, or calcified material on the inside of the artery. The build up of these deposits is called atherosclerosis. Atherosclerosis reduces the blood flow to the muscle of the heart or legs, which starves the muscle of oxygen, leading to either/or angina pectoris (chest pain), myocardial infarction (heart attack), and congestive heart failure, as the disease involves arteries supplying the heart, or pain in the leg (claudication) or leg ulcers if the disease involves arteries supplying the leg .

The body has natural mechanisms whereby new blood vessels, known as collaterals, grow to bypass arterial blockages, although these collaterals rarely are sufficient to restore blood flow to normal. Small narrow collateral blood vessels normally are present, connecting with the large blood vessels that carry the bulk of blood flow, but are too narrow to carry much blood flow under normal conditions. However, after the large vessels to which the collaterals connect become obstructed with atherosclerotic plaque, the collaterals can enlarge so that they are capable of delivering blood to the tissues originally supplied by the now obstructed vessel.

The use of recombinant genes or growth-factors to enhance myocardial collateral blood vessel function represents a new approach to the treatment of cardiovascular disease. Kornowski, R., et al., "Delivery strategies for therapeutic myocardial angiogenesis", *Circulation* 2000; 101:454-458. Proof of concept has been demonstrated in animal models of myocardial ischemia, and clinical trials are underway. Unger, E.F., et al., "Basic fibroblast

growth factor enhances myocardial collateral flow in a canine model", *Am J Physiol* 1994; 266:H1588-1595; Banai, S. et al., "Angiogenic-induced enhancement of collateral blood flow to ischemic myocardium by vascular endothelial growth factor in dogs", *Circulation* 1994; 83-2189; Lazarous, D.F., et al., "Effect of chronic systemic administration of basic fibroblast growth factor on collateral development in the canine heart", *Circulation* 1995; 91:145-153; Lazarous, D.F., et al., "Comparative effects of basic development and the arterial response to injury", *Circulation* 1996; 94:1074-1082; Giordano, F.J., et al., "Intracoronary gene transfer of fibroblast growth factor-5 increases blood flow and contractile function in an ischemic region of the heart", *Nature Med* 1996; 2:534-9.

Despite the promising hope for therapeutic angiogenesis as a new modality to treat patients with coronary artery disease, it is apparent that new strategies for optimally promoting clinically relevant therapeutic angiogenic responses are greatly to be desired. In particular, Moreover, new and improved angiogenesis strategies cause functionally that can cause relevant improvement in blood flow to an affected tissue are greatly desirable.

Summary of the Invention

The present invention overcomes the problems and disadvantages associated with current strategies and designs and provides kits, compositions and methods for angiotyping" individual patients to predict the likelihood of whether a given individual will develop good vs. poor collaterals naturally.

Several animal studies suggest that factors may exist that interfere with collateral growth--these include diabetes and hypercholesterolemia. There are subgroups of patients with coronary artery disease who have poor collaterals, and others who have excellent collaterals. Impaired collateral development occurring in response to arterial obstructive disease, or in response to angiogenesis interventions, is determined to a large extent by genetic factors (such as specific genetic polymorphisms), and/or by epigenetic factors (such as DNA methylation patterns) that alter the expression of genes encoding angiogenesis factors. Because of the marked individual variability that exists in the capacity to develop collaterals, and because such individual variability is based in large part on genetic and epigenetic differences among patients, it is important to be able to diagnose whether 1) a given patient is likely to develop good vs. poor collaterals naturally, and 2) a given patient is likely to respond to a specific therapeutic angiogenesis strategy. Because of these individual differences, angiogenesis treatment can ultimately be tailored to the individual patient.

Therefore, the present invention permits, through DNA and/or protein expression profiling using DNA chips or similar technology, diagnostic "angiotyping" of individual patients to predict the likelihood of whether a given individual will develop good vs. poor collaterals naturally, or in response to specific angiogenesis therapy.

One embodiment of the invention is directed to methods for "angiotyping" individual patients to predict the likelihood of whether a given individual will develop good vs. poor collaterals naturally. Accordingly, this can involve obtaining and providing a list of genes involved in collateral development.

Another embodiment of the invention is directed to methods for "angiotyping" individual patients to predict the likelihood of whether a given individual will develop good vs. poor collaterals in response to specific angiogenesis therapy.

Another embodiment of the invention is directed to methods for the detection of good vs. poor collaterals, comprising the detection of single nucleotide polymorphisms (SNPs) of an array of genes that have been determined through our experimental studies as being differentially expressed in tissues in which collaterals are developing in response to arterial occlusion. SNPs are detected using microchips or similar technology assaying for all, or most, of the genes determined to play a role in collateral development. The presence of a predisposition to develop poor vs. good collaterals is indicated by the presence of SNPs involving one or more of the genes we have determined are involved in those processes leading to enhanced collateral development.

Another embodiment of the invention is directed to methods for the detection of good vs. poor collaterals, comprises the detection of alterations of proteins in the blood, for example in peripheral blood mononuclear cells, expressed by the array of genes that have been determined through our experimental studies as being differentially expressed in tissues in which collaterals are developing in response to arterial occlusion. Protein levels will be either higher than normal levels, lower than normal levels, or the proteins will be post-translationally modified, such as, but not limited to changes in phosphorylation states. The determination of such protein levels/modifications can be by standard assays of individual proteins (ELISA, etc), or by newer methods, such as proteomic analysis. The presence of a predisposition to develop poor vs. good collaterals is indicated by the presence of lower or higher blood levels of proteins that are encoded by one or more of the genes we have determined are involved in those processes leading to enhanced collateral development. The

levels of protein can be measured, for example, in the blood fluid and/or in blood cells, such as peripheral blood mononuclear cells (PBMCs).

Another embodiment of the invention is directed to methods for the detection of good vs. poor collaterals, and comprises the detection of DNA methylation patterns involving those genes that have been determined to be differentially expressed in tissues in which collaterals are developing in response to arterial occlusion. The presence of a predisposition to develop poor vs good collaterals is indicated by the presence of DNA methylation patterns that alter gene expression, resulting in lower or higher blood levels of proteins that are encoded by one or more of the genes we have determined are involved in those processes leading to enhanced collateral development.

Another embodiment of the invention is directed to kits suitable for performing genetic microarray analysis for detection, where the kit comprises reagents, such as nucleic acid arrays (gene ships) or PCR primer sets that can detect relevant SNPs of most or all of the genes that have been determined to be involved in those processes leading to enhanced collateral development. The genes may be selected from the group of genes listed in Table 1. The sample may comprise, lymph, venous or arterial blood, and/or vascular tissue of the individual. In one embodiment the polymorphisms are detected using a genetic microarray. In another embodiment the polymorphisms are detected using quantitative PCR.

Another embodiment of the invention is directed to kits for carrying out any of the methods described above.

In specific embodiments the invention provides a method for predicting the likelihood that a subject will develop collaterals, comprising assaying the expression level of at least three in genes in the subject. in a sample obtained from the mammal. The likelihood of collateral development may be predicted by the altered expression of at least three, at least five, at least ten, at least twenty genes, or at least twenty genes in the sample. The altered expression may be increased or decreased expression. Genes having increased and decreased expression are listed in Tables 2 and 3 respectively. The altered expression level may be at least two fold higher or lower than a reference level. The level of gene expression may be determined by assaying the level of protein expression in a sample. In each of these embodiments, the sample may contain blood from the subject and/or may contain blood cells, such as PBMCs, from the subject.

In other embodiments of the invention, there is provided a method for predicting the likelihood that a subject will develop collaterals, comprising detecting the presence of at least three genetic variations in a sample from the patient, where the genetic variations are SNPs or altered DNA methylation patterns. The likelihood of collateral development can be predicted by the presence of genetic variations in at least three, at least five, at least ten, at least twenty genes, or at least twenty genes in the sample. The genes may be selected from the group consisting of the genes listed in Table 1. The method of assay may comprise using a genetic microarray or quantitative PCR, and may be a method to detect DNA methylation patterns and/or to detect single nucleotide polymorphisms.

The invention also provides a kit for carrying out the assays described above, where the assay is to be carried out using a PCR and where the kit comprises a set of primers suitable for amplifying at least three, at least five, at least ten, or at least twenty DNA or RNA sequences corresponding to the genes in Table 1. In another example, there is provided a kit for carrying out the assays described above where the kit comprises a nucleic acid array capable of detecting single nucleotide polymorphisms in a plurality or majority of the genes identified in Table 1.

In another embodiment, the expression level of the genes may be determined by measuring the concentration of the proteins, for example, soluble proteins, encoded by the genes listed in Table 1. The sample from the subject may be blood, and/or lymph. The level of protein expressions may, for example, be determined by ELISA.

The invention also provides methods for promoting collateral formation in a subject, by administering to the subject a composition that decreases expression of at least one gene identified in Table 2 and/or that increases expression of at least one gene identified in Table 3. The composition may contain an antisense oligonucleotide, an siRNA molecule, an RNAi molecule, an oligonucleotide that binds to mRNA to form a triplex, or a DNA molecule that is transcribed in the subject to produce an antisense oligonucleotide, an siRNA molecule, an RNAi, or an oligonucleotide that binds to mRNA to form a triplex. The composition may contain an antibody or a soluble protein receptor, for example, a human antibody or a human soluble protein receptor, that binds to a protein that inhibits collateral formation in the subject. The composition may comprise a protein that is administered to supplement the loss of a protein encoded by a gene identified in Table 3.

Other objects, features and advantages of the present invention will become apparent from the following detailed description. It should be understood, however, that the detailed description and the specific examples, while indicating preferred embodiments of the invention, are given by way of illustration only, since various changes and modifications within the spirit and scope of the invention will become apparent to those skilled in the art from this detailed description.

Description of the Figures

Table 1 lists the genes whose expression was detectably altered during the development of collaterals.

Table 2 lists the genes whose expression was increased during the development of collaterals, and also shows the time course of the changes in gene expression.

Table 3 lists the genes whose expression was decreased during the development of collaterals, and also shows the time course of the changes in gene expression

Detailed Description of the Invention

The present invention provides kits, compositions and methods for angiotyping individual patients and for predicting the likelihood of whether a given individual will develop good vs. poor collaterals, either naturally or in response to specific angiogenesis therapy. Specifically, those genes that have altered expression levels during the development of collaterals have been identified, and the changes in gene expression have been quantified. By measuring changes in gene expression, the risk of whether a given individual will develop good vs. poor collaterals naturally or in response to specific angiogenesis therapy can be determined. Moreover, the relative changes in gene expression at different time points during the collateral development process have been measured, and these measurements allow additional insight into the progress and development of collaterals.

Because differential expression of genes is involved in collateral development, changes in the degree of expression, or in the length of time during which they are differentially expressed, lead to different degrees of collateral development. In the context of coronary artery disease and peripheral vascular disease, differing degrees of collateral development can cause some individuals to have minimal symptoms in association with atherosclerotic arterial obstructive disease, and other individuals to have severe symptoms.

Changes in the degree of gene expression, or in the length of time during which the genes are differentially expressed, are caused by polymorphisms either in the coding region of the gene or in the regulatory components of the gene. Alternatively, these changes can be caused by "epigenetic alterations," such as, but not limited to, changes in DNA methylation patterns. By correlating changes in gene expression with collateral development, the present invention identifies those genes in which polymorphisms or altered DNA methylation patterns can convey susceptibility to the development of either poor vs good collateral development.

The identification of genes that are involved in collateral development allows those genes having changed degree or duration of expression, caused in part by polymorphisms of the gene or alterations in DNA methylation patterns, to be used as targets to identify genetic abnormalities conveying altered capacities to develop collaterals. Identification of polymorphisms or alterations in DNA methylation patterns allows prediction of the risk for poor collateral development in patients prior to the performance of angioplasty procedures or the initiation of angiogenesis therapy. Once pre-procedure risk prediction is possible, this will importantly influence how a patient is treated. Some patients deemed to be resistant to the development of collaterals might be offered bypass surgery or angioplasty. Others might forego angiogenesis therapy and be treated aggressively with brachytherapy (intravascular radiation). Accordingly, the present invention provides new and improved methods for "angiotyping" individual patients to predict the likelihood of whether a given individual will develop good vs poor collaterals naturally or in response to specific angiogenesis therapy.

Moreover, identification of the genes that are abnormally expressed by an individual patient because of either a SNP or an altered DNA methylation pattern, provides new methods for ameliorating or treating the disease by therapy targeted to a specific set or subset of those genes with altered expression. Because different polymorphisms and DNA methylation patterns play a role in the development of collaterals in different patients, the invention allows identification of specific abnormalities that may be characteristic to a specific patient. The invention therefore allows for greater specificity of treatment. A regime that may be efficacious in one patient with a specific polymorphism profile may not be effective in a second patient with a different polymorphism profile. Such profiling also allows treatment to be individualized so that unnecessary side effects of a treatment strategy that would not be effective for a specific patient can be avoided.

Specifically, approximately five hundred and seventy five genes are identified whose expression changes during the course of collateral development. Since the differential expression of these genes is involved in collateral development, changes in the degree of expression, or in the length of time during which they are differentially expressed, leads to altered capacity to develop collaterals.

Changes in the degree of gene expression, or in the length of time during which the genes are differentially expressed, can be caused by polymorphisms in the gene or in the regulatory components of the gene. Such polymorphisms, conveying an increased risk of disease development, have already been identified for several genes associated with several diseases. This invention, therefore, identifies those genes in which polymorphisms can convey susceptibility to poor vs good collateral development. Similar predictions can derive from altered gene expression caused by altered DNA methylation patterns, which can relate to specific SNPs, or regulate gene expression independently of SNPs. Subsequent reference, therefore, to prediction of good vs poor collateral development, relate to polymorphisms of the genes identified by this invention, or of their regulatory units, or to altered DNA methylation patterns which in turn alter gene expression.

The change in expression of certain of the identified genes is predictive of the capacity to develop poor vs. good collaterals. By identifying 575 genes whose expression changes during collateral development, the inventors recognize that analysis of greater numbers of polymorphisms or DNA methylation patterns of those genes leads to a greater ability to predict the capacity to develop collaterals. The role played by these genes in collateral development means that an ability to manipulate the expression of those genes permits improved treatment of arterial obstructive disease . The skilled artisan will recognize that methods to enhance or decrease gene expression are known in the art. For example, methods to enhance collaterals may include gene therapy to increase the expression of genes down-regulated during collateral development. Such gene therapy can be carried out using methods that are known in the art and can used, for example, viral and/or non-viral vectors to deliver nucleic acids that encode and permit expression of a desired gene. Conversely, methods of decreasing expression and/or activity of a desired gene are well known in the art and include, for example, antisense RNA, and RNAi/siRNA methods. Treatment may also include methods to decrease the expression of genes up-regulated during collateral development.

Identification of genes involved in collateral development also permits identification of proteins that affect the development of collaterals. This in turn makes possible the use of methods to expression of these proteins or alter their metabolism. Methods to alter the effect of expressed proteins include, but are not limited to, the use of specific antibodies or antibody fragments that bind the identified proteins, specific receptors and soluble receptor fragments that bind the identified protein, or other ligands or small molecules that inhibit the identified protein from affecting its physiological target and exerting its metabolic and biologic effects. In addition, those proteins that are down-regulated during the course of collateral development may be supplemented exogenously to ameliorate their decreased synthesis.

Different polymorphisms and DNA methylation patterns may play a role in collateral development in different patients. Accordingly, the present invention makes possible an identification of specific abnormalities that are characteristic of a specific patient ("angiotyping"), which allows for greater specificity of treatment. A regime that may be efficacious in one patient with a specific polymorphism profile may not be effective in a second patient with a different polymorphism profile. Such a profiling also allows treatment to be individualized so that unnecessary side effects of a treatment strategy that would not be effective for a specific patient can be avoided.

Elucidation of Changes in Gene Expression in Collateral Development

The inventors have identified the genes that undergo changes in expression during collateral development. Those genes are listed in Table 1. Those genes that exhibit increased and decreased expression during collateral development are shown in Tables 2 and 3 respectively, together with measurements of the temporal changes in expression. The inventors have carried out this analysis using nucleic acid array analysis of murine adductor muscles as described in more detail below. The skilled artisan will recognize, however, that additional methods for measuring gene expression are well known in the art.

The mouse is a widely accepted model for the human for vascular studies, and results obtained in the mouse are considered highly predictive of results in humans. Accordingly, it is expected that the changes in gene expression in humans during collateral development will be similar to or essentially the same as those observed in the mouse. Exaggerated changes in the degree of expression in these genes, or in the length of time during which the genes are differentially expressed, will predispose to good vs poor collaterals. Such exaggerated changes are usually caused by polymorphisms in the gene or in the regulatory components of

the gene, and therefore the mouse genes identified as being differentially regulated during the angiogenic process will be homologous to the human genes in which such polymorphisms will be found to convey the ability to form good vs. poor collaterals. Moreover, both mouse and human homologues are known for each of the genes described in Table 1, demonstrating further that the results obtained in the mouse studies will be highly predictive of results obtained in humans.

The genes for which, in a given patient, either SNPs or altered DNA methylation patterns are observed, and that are associated with collateral development, also serve as the target for therapeutic interventions. Thus, those genes upregulated during the collateral development can be targeted by therapy designed to decrease gene expression or function of the proteins encoded by these genes; and those genes down-regulated during collateral development can be targeted by therapy designed to increase gene expression or function of the proteins encoded by these genes.

Changes in gene expression in the mouse ischemic hindlimb during experimentally induced collateral development have been studied, a model commonly accepted as a reasonable animal model simulating collateral development as it occurs in humans. Sample and control mouse hindlimb tissues were obtained, RNA was prepared from the tissues, labeled cRNA generated from it and analyzed using an Affymetrix GeneChip® mouse Genome. Sample and control tissues were compared and those genes that experienced significant changes in gene expression were identified. For the purposes of this study, a two fold increase or decrease in gene expression was deemed significant, although the skilled worker will recognize that under certain circumstances smaller changes in gene expression may also be significant. Corresponding human genes for each of the genes determined to have a significant change in expression were identified.

Although about 575 genes have been shown to have altered expression in collateral development (Table 1), it is possible to reliably predict good vs poor collateral development by analyzing a subset of a few of these genes. In embodiments of the present invention at least five, ten, fifteen, twenty or fifty genes may be studied or, if desired, all or most of the genes listed in Table 1 can be studied. These genes also can be analyzed for polymorphisms or altered DNA methylation patterns that alter gene expression. All of the genes can be analyzed initially, but reliable predictions can be made by analyzing a subset of these genes that contains a few members. In other embodiments, at least five, ten, fifteen, twenty or fifty

genes may be studied or, if desired, all or most of the genes listed in Table 1 can be studied, for example, using sequencing, short tandem repeat association studies, single nucleotide polymorphism association studies, etc. In each case, however, it generally is more convenient to study gene expression or polymorphisms in a smaller subset of the genes.

By measuring changes in expression of a set of genes (for example by blood protein analysis or by analysis of proteins in blood cells such as PBMCs), or by identification of polymorphisms or DNA methylation patterns influencing expression of sets of genes, rather than of a single gene, the present invention provides increased statistical confidence that the changes observed are predictive of poor vs. good collateral development, such as by providing reliable risk profiling of an individual. Thus, a change in expression of a single gene, or a single gene polymorphism, may not increase susceptibility to good vs poor collateral development sufficiently to cross the diagnosis threshold. On the other hand, coordinated changes in expression of multiple specified genes, due the presence of multiple polymorphisms and/or DNA methylation patterns, are much more likely to increase the likelihood of poor vs. good collateral development. This is analogous to the situation of an individual have only one risk factor predisposing to atherosclerosis (elevated cholesterol). Risk is increased markedly as the number of risk factors increase (elevated cholesterol plus hypertension, obesity, smoking, diabetes, etc).

Identification of polymorphisms or alterations in DNA methylation patterns allows prediction of the risk for poor collateral development in patients prior to the performance of angioplasty procedures or the initiation of angiogenesis therapy. This pre-procedure risk prediction can be used to influence how the patient is treated. Some patients deemed to be resistant to the development of collaterals might be offered bypass surgery or angioplasty. Others might forego angiogenesis therapy and be treated aggressively with brachytherapy (intravascular radiation). Accordingly, the present invention provides new and improved methods for "angiotyping" individual patients to predict the likelihood of whether a given individual will develop good vs poor collaterals naturally or in response to specific angiogenesis therapy.

Dysregulation of Multiple Genes that Increase Susceptibility to Poor vs Good Collateral Development

Gene polymorphisms and altered DNA methylation patterns that lead to biologically important alterations in the expression of genes that are differentially expressed during

collateral development can be measured directly in patient samples. These samples comprise DNA that is most conveniently obtained from peripheral blood, for example from PBMCs. The present inventors used nucleic acid array methods to identify the complete set of genes that exhibit significantly changed expression during the course of the healing response to acute vascular injury. However, other methods for measuring changes in gene expression are well known in the art. For example, levels of proteins can be measured in tissue sample isolates using quantitative immunoassays such as the ELISA. Kits for measuring levels of many proteins using ELISA methods are commercially available from suppliers such as R&D Systems (Minneapolis, MN) and ELISA methods also can be developed using well known techniques. See for example Antibodies: A Laboratory Manual (Harlow and Lane Eds. Cold Spring Harbor Press). Antibodies for use in such ELISA methods either are commercially available or may be prepared using well known methods.

Other methods of quantitative analysis of multiple proteins include, for example, proteomics technologies such as isotope coded affinity tag reagents, MALDI TOF/TOF tandem mass spectrometry, and 2D-gel/mass spectrometry technologies. These technologies are commercially available from, for example, Large Scale Proteomics Inc. (Germantown MD) and Oxford Glycosystems (Oxford UK).

Alternatively, quantitative mRNA amplification methods, such as quantitative RT-PCR, can be used to measure changes in gene expression at the message level. Systems for carrying out these methods also are commercially available, for example the TaqMan system (Roche Molecular System, Alameda, CA) and the Light Cycler system (Roche Diagnostics, Indianapolis, IN). Methods for devising appropriate primers for use in RT-PCR and related methods are well known in the art. In particular, a number of software packages are commercially available for devising PCR primer sequences.

Nucleic acid arrays offer are a particularly attractive method for studying the expression of multiple genes. In particular, arrays provide a method of simultaneously assaying expression of a large number of genes. Such methods are now well known in the art and commercial systems are available from, for example, Affymetrix (Santa Clara, CA), Incyte (Palo Alto, CA), Research Genetics (Huntsville, AL) and Agilent (Palo Alto, CA). See also US Patent Nos. 5,445,934, 5,700,637, 6,080,585, 6,261,776 which are hereby incorporated by reference in their entirety.

Changes in the degree of gene expression, or in the length of time during which the genes are differentially expressed, can be caused by polymorphisms in the gene or in the regulatory components of the gene. Such polymorphisms, conveying an increased risk of disease development, have already been identified for genes associated with several diseases. The present invention, therefore, identifies those genes in which polymorphisms or altered DNA methylation patterns can convey susceptibility to poor vs good collateral development. It is one object of this invention to identify such polymorphisms by developing a DNA microarray chip containing all those SNPs affecting those genes we have identified as playing a role in collateral development (For example, by using the Affymetrix GeneChip system).

Methods for identifying polymorphisms in genes are well known in the art. See, for example, United States Patent Nos. 6,235,480 and 6,268,146, which are hereby incorporated by reference in their entirety. Once polymorphisms are identified, methods for detecting specific polymorphisms in a gene using nucleic acid arrays are also well known in the art

Thus, in one embodiment, the invention provides methods where SNPs or altered DNA methylation patterns are identified for at least three genes selected from the genes shown in Table 1. In other embodiments of the invention SNPs or altered DNA methylation patterns are determined of at least five genes to determine the likelihood of good vs poor collateral development. In yet further embodiments the number of genes assayed is ten. In yet other embodiments the number of genes assayed is 20 or at least about 20. In still yet other embodiments the number of genes assayed is 50 or at least about 50. Regardless of the number of genes in the subset of analyzed genes, selected from the genes shown in Table 1, the aggregate number of polymorphisms or DNA methylation patterns can then permit prediction of good vs poor collateral development. Similarly, coordinated changes in expression of the genes identified herein also can permit prediction of good vs poor collateral development.

With respect to polymorphisms, as the number of biologically significant polymorphisms increases, so does the confidence of the predictions that can be made. Similarly, coordinated changes in expression of a greater number of the identified genes indicates increases the confidence with which predictions can be made. As more polymorphisms of the genes listed in Table 1 are identified, even more powerful risk profiling will be possible. Thus, in other embodiments of the invention the expression of at least five genes or at least about five genes is assayed to determine the capacity of collateral

development. In yet further embodiments the number of genes assayed is ten. In yet other embodiments the number of genes assayed is 20 or at least about 20. In still yet other embodiments the number of genes assayed is 50 or at least about 50.

The skilled artisan will recognize that, due to the heterogeneous nature of collateral development, not all individuals with poor collateral development will exhibit altered expression of every last one of the genes listed in Table 1. Thus, it is possible that one, a few, or many genes will not exhibit significantly altered expression (and therefore will contain no biologically important polymorphisms or altered DNA methylation patterns), and that different individuals will exhibit different combinations; yet, the coordinated changes induced by the polymorphisms in the expression of the totality of genes are highly predictive of the presence of prediction of poor vs good collateral development.

In general, where the expression of only a relatively small number of genes is studied, changes in expression in most or all of the genes can be observed to provide a reliable diagnosis of good vs poor collateral development. For example, where only three genes are measured, all three genes can show relevant changes in expression to permit a reliable diagnosis impaired collateral development. Where five genes are studied, changes in at least four genes typically will provide a reliable diagnosis. Where ten genes are measured, a reliable diagnosis is obtained where changes in at least seven genes are observed. Where more than 10 genes are measured, changes in 90%, 80%, 70%, 60% or 50% of the measured genes are predictive of impaired collateral development. As these percentages decrease, the reliability of the diagnosis also decreases, but the skilled worker will recognize that when a coordinated change in expression of 20 or 30 genes of the genes listed in Table 1 is observed this is highly predictive of the likelihood of poor vs good collateral development. In general, as the number of genes increases, it is possible to provide a reliable diagnosis by observing coordinated changes in expression in a relatively smaller subset of the genes studied.

Tissues Sampled to Determine Altered Gene Expression and the Presence of Polymorphisms that Cause Biologically Important Alterations in Relevant Gene Expression

Although any sample containing nucleic acid would be appropriate for this purpose, the simplest tissue to sample is peripheral venous or arterial blood. However, other tissues may be used, such as vascular tissue, in particular arterial vascular tissue or venous vascular tissue.

Methods of Studying Gene Polymorphisms, DNA methylation patterns, and protein levels of the Genes Listed in Table 1

Polymorphisms can be identified by several methods including restriction enzyme digestion, sequencing, short tandem repeat association studies, single nucleotide polymorphism association studies, etc. These methods are well-known in the art.

Gene expression can also be studied at the protein level. Target tissue is first isolated and then total protein is extracted by well known methods. Quantitative analysis is achieved, for example, using ELISA methods employing a pair of antibodies specific to the target protein(s).

A subset of the proteins listed in Table 1 are soluble or secreted. In such instances the proteins may be found in the blood, plasma or lymph and an analysis of those proteins may be afforded by any of those methods described for the analysis of proteins in such tissues. This provides a minimally invasive means of obtaining patient samples for predicting the ability to generate collaterals. Methods for identifying secreted proteins are known in the art.

Gene polymorphisms are detected reliably with tissue derived from any source, including peripheral blood; blood protein levels can serve as a source of identifying altered gene expression.

RNA Expression

Methods of isolating RNA from tissue are well known in the art. See, for example, Sambrook *et al. Molecular Cloning: A Laboratory Manual (Third Edition)* Cold Spring Harbor Press, 2001. Commercial reagents also are available for isolating RNA.

Briefly, for example, cells or tissue are lysed and the lysed cells centrifuged to remove the nuclear pellet. The supernatant is then recovered and the nucleic acid extracted using phenol/chloroform extraction followed by ethanol precipitation. This provides total RNA, which can be quantified by measurement of optical density at 260-280 nM.

mRNA can be isolated from total RNA by exploiting the "PolyA" tail of mRNA by use of several commercially available kits. QIAGEN mRNA Midi kit (Cat. No. 70042); Promega PolyATtract® mRNA Isolation Systems (Cat. No. Z5200). The QIAGEN kit provides a spin column using Oligotex Resin designed for the isolation of poly A mRNA and yields essentially pure mRNA from total RNA within 30 minutes. The Promega system uses a biotinylated oligo dT probe to hybridize to the mRNA poly A tail and requires about 45 minutes to isolate pure mRNA.

mRNA can also be isolated by using the cesium chloride cushion gradient method. Briefly the flash frozen tissue if homogenized in Guanethedium isothiocyanate, layered over a cushion of cesium chloride and ultracentrifuged for 24 hours to obtain the total RNA.

Genetic Microarray Analysis

Microarray technology is an extremely powerful method for assaying the expression of multiple genes in a single sample of mRNA. For example, Gene Chip® technology commercially available from Affymetrix Inc. (Santa Clara, Ca) uses a chip that is that is plated with probes for over thousands of known genes and expressed sequence tags (ESTs). Biotinylated cRNA (linearly amplified RNA) is prepared and hybridized to the probes on the chip. Complementary sequences are then visualized and the intensity of the signal is commensurate with the number of copies of mRNA expressed by the gene.

Protein Expression

Gene expression may also be studied at the protein level. Target tissue is first isolated and then total protein is extracted by well known methods. Quantitative analysis is achieved, for example, using ELISA methods employing a pair of antibodies specific to the target protein.

A subset of the proteins listed in Table 1 are soluble or secreted. In such instances the proteins may be found in the blood, plasma or lymph and an analysis of those proteins may be afforded by any of those methods described for the analysis of proteins in such tissues. This provides a minimally invasive means of obtaining patient samples for estimate of risk of developing restenosis or of atherosclerosis. Methods for identifying secreted proteins are known in the art.

The emerging technology of proteomics can supply a powerful analytic tool to assay for changes in large numbers of proteins.

The following examples are offered to illustrate embodiments of the present invention, but should not be viewed as limiting the scope of the invention.

Examples

Microarray Analysis of the Mouse Hindlimb

Isolation of RNA

Mice underwent femoral artery ligation and extirpation.A control group was treated by sham surgery. Mouse adductor muscles after surgery and sham surgery were collected

and flash frozen. Pooled muscles (30-50mg) were crushed into powder using a mortar and pestle (collected with liquid nitrogen) and then homogenized in 2.5 ml of guanidinium isothiocyanate. Total RNA was extracted using ultracentrifugation on cesium chloride cushion gradient for 24 hours at 4°C. See Sambrook et al *supra*.

Target Preparation and DNA Microarray Hybridizations

For the first strand cDNA synthesis reaction, 5.0-8.0 μg of total RNA was incubated at 70°C for 10 minutes with T7-(dT) 24 primer, then placed on ice. For the temperature adjustment step, 5X first stand cDNA buffer, 0.1 M DTT, and 10 mM dNTP mix was added and the reaction incubated for 1 hour at 42°C. SSII reverse transcriptase was added, and the reaction incubated for 1 hour at 42°C. With the first strand synthesis completed, 5X second strand reaction buffer, 10 mM dATP, dCTP, dGTP, dTTP, DNA Ligase, DNA Polymerase I, and RNaseH were added to the reaction tube. Samples were then incubated at 16 °. Following the addition of 0.5M EDTA, cDNA was cleaned using phase lock gels-phenol/chloroform extraction, followed by ethanol precipitation.

Synthesis of Biotin-Labeled cRNA (*In vitro* transcription)

The synthesis of biotin-labeled cRNA was completed using the ENZO BioArray RNA transcript labeling kit from (ENZO Biochem, Inc., New York, NY) according to the manufacturers protocol. To set up the reaction 1 μg of cDNA, 10X HY reaction buffer, 10X Biotin labeled ribonucleotides, 10X DTT, 10X RNase inhibitor mix and 20X T7 RNA polymerase were incubated at 37°C for 4-5 hours. RNeasy spin columns from QIAGEN were used to purify the labeled RNA, followed by ethanol precipitation and quantification.

Fragmentation of cRNA for Target Preparation

5X fragmentation buffer (200 mM Tris-acetate, pH 8.1, 500 mM KOAc, 150 mM Mg)Ac) was added to the cRNA. Samples were incubated at 94°C for 35 minutes, then placed on ice. Fragmented cRNA was stored at –70°C.

Target Hybridization

Hybridization cocktail was prepared as follows: fragmented cRNA (15 μg adjusted), control oligonucleotide B2 (Affymetrix), 20X eukaryotic hybridization controls (Affymetrix), herring sperm DNA, acetylated BSA, and 2X hybridization buffer (Affymetrix) were combined, and heated to 99°C for five minutes. Hybridization cocktail was then centrifuged at maximum speed for five minutes to remove any insoluble materials from the mixture.

Following centrifugation, cocktail was heated at 45°C for five minutes. The clarified hybridization cocktail was then added to the Affymetrix probe array cartridge that had been pre-wet with 1X hybridization buffer. The probe array was then placed in a 45°C rotisserie box oven set at 60 rpm and hybridized for 16 hours.

Washing, Staining and Scanning Probe Arrays

The GeneChip® Fluidics Station 400 was used to wash and stain the array. This instrument was run using GeneChip® software. Briefly, arrays were washed for 10 cycles with non-stringent wash buffer at 25°C, followed by 4 cycles of washing with stringent wash buffer at 50°C. The array was then stained for 10 minutes with Phycoerythrin-streptavidin at 25°C. The array was then washed for 10 cycles with non-stringent wash buffer at 25°C. The probe array was the stained again with phycoerythrin-streptavidin for 10 minutes at 25°C, and then washed for 15 cycles with non-stringent wash buffer at 30°C. Hybridization signals are detected by placing the probe array in an HP Gene Array™ Scanner, which operated using GeneChip® software.

Data Analysis

Data analysis was performed using GeneChip® software (version 3.3) using the manufacturer's instructions. Lockhart, D.J. *et al.*, Nat. Biotechnol. 14:1675-80 (1996). Briefly, each gene was represented and queried by 1-3 probe sets on the chip. Each probe set comprises 16 perfect match (PM) and 16 mismatch (MM) 25 nucleotide base probes. The mismatch has a single base change in the middle of the 25 base pair probe. The hybridization signal from the PM and the MM probes were compared and this allowed for a measure of signal intensity that is specific and eliminated the nonspecific cross hybridization from the data of the two control chips. Intensity differences as well as ratios of intensity of each probe pair are used to make a "present" or "absent" call. The controls were used as baseline and the experimental GeneChip® assay values compared to the base line to derive four matrixes which were used to determine the difference calls that indicate whether the transcription level of a particular gene is changed.

Iterative comparisons were performed using a spreadsheet analysis (Microsoft Excel). Each experimental data set at a particular time point (n=2) and the difference in expression between the controls and experimental was determined for each gene. Genes with a consistent difference call across all four pairwise comparisons were extracted for further analysis.

GeneSpring® Analysis

The data from each GeneChip® assay was fed into the GeneSpring® software and clustering of genes based on their temporal expression profile was analyzed. Correlation coefficients of 0.97 or greater were taken as a cutoff to create gene-clusters with significant expression homology.

Other embodiments and uses of the invention will be apparent to those skilled in the art from consideration of the specification and practice of the invention disclosed herein. All references cited herein, including all U.S. and foreign patents and patent applications, are specifically and entirely hereby incorporated herein by reference. It is intended that the specification and examples be considered exemplary only, with the true scope and spirit of the invention indicated by the following claims.

Claims

What is claimed is:

1. A method for predicting the likelihood that a subject will develop collaterals, comprising assaying the expression level of at least three in genes in said subject. in a sample obtained from said mammal.

2. The method according to claim 1, wherein the likelihood of collateral development is predicted by the altered expression of at least three, at least five, at least ten, at least twenty genes, or at least twenty genes in said sample.

3. The method according to claim 1, wherein the likelihood of collateral development is predicted by increased expression of at least three, at least five, at least ten, at least twenty genes, or at least twenty genes in said sample.

4. The method according to claim 1, wherein the likelihood of collateral development is predicted by decreased expression of at least three, at least five, at least ten, at least twenty genes, or at least twenty genes in said sample.

5. The method according to claim 1 or claim 2, wherein said genes are selected from the genes listed in Table 1.

6. The method according to claim 3, wherein said genes are selected from the genes listed in Table 2.

7. The method according to claim 4, wherein said genes are selected from the genes listed in Table 2.

8. The method according to claim 1 wherein said sample comprises blood from said subject.

9. The method according to claim 1, wherein said altered expression level is at least two fold higher or lower than a reference level.

10. The method of any of claims 1-9 wherein the level of gene expression is determined by assaying the level of protein expression in a sample.

11. A method for predicting the likelihood that a subject will develop collaterals, comprising detecting the presence of at least three genetic variations in a sample from said patient, wherein said genetic variations are SNPs or altered DNA methylation patterns..

12. The method according to claim 11, wherein the likelihood of collateral development is predicted by the presence of genetic variations in at least three, at least five, at least ten, at least twenty genes, or at least twenty genes in said sample.

13. The method according to claim 11 or 12, wherein said genes are selected from the group consisting of the genes listed in Table 1.

14. The method according to claim 1 or claim 11 wherein the method of assay comprises using a genetic microarray or quantitative PCR..

15. The method according to claim 11 wherein the assay comprises a method to detect DNA methylation patterns.

16. The method according to claim 11 wherein the assay comprises a method to detect single nucleotide polymorphisms.

17. A kit for carrying out the assay according to claim 1 or claim 11, wherein said assay is to be carried out using a PCR and wherein said kit comprises a set of primers suitable for amplifying at least three, at least five, at least ten, or at least twenty DNA or RNA sequences corresponding to the genes in Table 1.

18. A kit for carrying out the assay according to claim 11 wherein said kit comprises a nucleic acid array capable of detecting single nucleotide polymorphisms in a plurality of the genes identified in Table 1.

19. A kit according to claim 18 wherein said array is capable of detecting single nucleotide polymorphisms, if present, in a majority of the genes identified in Table 1.

20. The method according to claim 1, wherein the expression level of said genes is determined by measuring the concentration of the proteins encoded by said genes.

21. The method according to claim 20, wherein said proteins are soluble proteins.

22. The method according to claim 21, wherein said sample is blood and/or lymph.

23. The method according to claim 20, wherein the level of protein expressions is determined by ELISA.

24. A method of promoting collateral formation in a subject, comprising administering to said subject a composition that decreases expression of at least one gene identified in Table 2 and/or that increases expression of at least one gene identified in Table 3.

25. The method according to claim 24, wherein said composition comprises an antisense oligonucleotide, an siRNA molecule, an RNAi molecule, an oligonucleotide that binds to mRNA to form a triplex, or a DNA molecule that is transcribed in said subject to produce an antisense oligonucleotide, an siRNA molecule, an RNAi, or an oligonucleotide that binds to mRNA to form a triplex.

26. The method according to claim 24, wherein said composition comprises an antibody or a soluble protein receptor that binds to a protein that inhibits collateral formation in said subject.

27. The method according to claim 26, wherein said composition comprises a human antibody or a human soluble protein receptor.

28. The method according to claim 24, wherein said composition comprises a protein that is administered to supplement the loss of a protein encoded by a gene identified in Table 3.

TABLE 1

Gene	Genebank #	Product
Fos	V00727	FBJ osteosarcoma oncogene
Timp	V00755	
Rrad	AF084466	Ras-like GTP-binding protein Rad
Scya7	X70058	cytokine
Snk	M96163	
Gp49b	U05265	gp49B2; gp49B1
Tc10l-pending	AW121127	
Krox-24	M28845	zinc finger protein
H3f3b	X13605	H3 histone, family 3B
Emp1	X98471	epithelial membrane protein-1
Alrp	AF041847	cardiac ankyrin repeat protein MCARP
THBS1	M62470	thrombospondin
Scya2	M19681	platelet-derived growth factor-inducible protein
Angptl4	AI326963	
gp49	M65027	cell surface antigen
rrg	D10837	lysyl oxidase
Cdkn1a	AW048937	cyclin-dependent kinase inhibitor 1A (P21)
Litaf-pending	AI852632	
mts1	M36579	
Lgals3	X16834	
Cmkbr5	AV370035	
c-myc	L00039	myelocytomatosis oncogene
Mknk2	Y11092	map kinase interacting kinase
Saa3	X03505	SAA3
Cyr61	M32490	cysteine rich protein 61
pgM	D45889	PG-M core protein
Cish3	U88328	suppressor of cytokine signalling-3
C5aR	S46665	C5a anaphylatoxin receptor
Mt2	K02236	
Zfp36	X14678	zinc finger protein 36
Scya9	U49513	macrophage inflammatory protein-1gamma
Spp1	X13986	secreted phosphoprotein 1
Atf3	U19118	LRG-21
Cd14	X13333	leucine-rich preprotein (AA -15 to 351)
Pde6a	X60664	rod phosphodiesterase alpha subunit

TABLE 1

Mmp3	X66402	stromelysin-i
Lgmn	AJ000990	legumain
C87222	AI836322	
Csf1r	X06368	colony stimulating factor 1 receptor
Cmkbr2	U56819	mcp-1 receptor
Lzm, Lzp, Lys	M21050	lysozyme M
Tdag	U44088	TDAG51
Cyp1b1	X78445	cytochrome P450EF B1
Slfn4	AF099977	schlafen4
E161	X61450	E161
Runx2	AV245229	
Tnc	X56304	precursor tenascin protein
Il17r	U31993	interleukin 17 receptor
S100a10	M16465	calcium binding protein A11 (calgizzarin)
	C85523	
Gro1	J04596	GRO1 oncogene
Pira3	U96684	PIRA3
Itgb2	M31039	complement receptor C3 beta-subunit
Evi2	M34896	ecotropic viral integration site 2
Cish3	AV374868	
Hmox1	X56824	haem oxygenase
Col3a1	AA655199	
Ugdh	AF061017	UDP-glucose dehydrogenase
Tyrobp	AF024637	DAP12
2610024P12Rik	AW124113	
Mt1	V00835	Metallothionein-I
Ywhag	AF058799	14-3-3 protein gamma
Cd68	X68273	macrosialin
Lzp-s	X51547	P lysozyme structural
Fcgr2b	M31312	Fc receptor, IgG, low affinity IIb
Crp2, SmLim	D88792	double LIM protein-1
OTS-8	M73748	glycoprotein 38
TSC-36	M91380	TGF-beta-inducible protein
Mpg-1	L20315	MPS1 protein
Lcn2	X81627	lipocalin
Fkbp10	L07063	FKBP65 binding protein

TABLE 1

Col3a1	AV234303	
Anxa1	AV003419	
Gfpt2	AB016780	Glutamine:fructose-6-phosphate amidotransferase 2
spi2/eb4	M64086	spi2 proteinase inhibitor
Thbd	X14432	thrombomodulin
5730470C09Rik	AA738776	
MRP8	M83218	intracellular calcium-binding protein
2310057H16Rik	AW215736	
Man1a	U04299	mannosyl-oligosaccharide alpha-1,2-mannosidase
Oaz1	AV212241	
Adam19	AA726223	
D15Wsu122e	AW123921	
Mlp	X61399	MARCKS-like protein
Sat	L10244	spermidine/spermine N1-acetyltransferase
Col3a1	X52046	type III collagen
mPHLL2	AB003433	photolyase/blue-light receptor homolog2
	AW047237	
	AI843046	
Angptl4	AA797604	
C1qb	M22531	complement component 1, q subcomponent, beta polypeptide
Apoe	D00466	apolipoprotein
Col14a1	AJ131395	collagen type XIV
Mail-pending	AA614971	
Ftl, Ftl-1	L39879	ferritin L-subunit
Ugt1a6	U16818	UDP glucuronosyltransferase
C1qa	X58861	complement subcomponent C1Q A-chain precursor
Ctss	AJ223208	cathepsin S
1600023E10Rik	AI849082	
2510002C21Rik	AA596710	
Col5a-2	L02918	procollagen type V alpha 2
Scya8	AB023418	monocyte chemoattractant protein-2 (MCP-2) precursor
AI035637	AI842259	
osf-2	D13664	osteoblast specific factor 2 precursor
Eln	U08210	tropoelastin
Stat5b	U21110	mammary gland factor
C1qc	X66295	C1q C chain

TABLE 1

Myh8	M12289	
Tubb5	X04663	tubulin, beta 5
PAI-1	M33960	plasminogen activator inhibitor
metalloelastase	M82831	metalloelastase
Vcl	L18880	vinculin
Sfrp2	U88567	secreted frizzled related protein sFRP-2
Bmk, Hck-1	J03023	hemopoietic cell kinase
Atp1b2	X16645	ATPase, Na+/K+ transporting, beta 2 polypeptide
Slpi	AF002719	secretory leukoprotease inhibitor
Tgif	X89749	mTGIF protein
Gbas	AJ001261	NIPSNAP2 protein
Fgfrp	U04204	aldose reductase-related protein
Anxa4	U72941	annexin IV
Gadd45a	U00937	GADD45 protein
Myf6	X59060	myogenic factor 6 (herculin)
Ext1	X96639	exostoses (multiple) 1
Mrc1	Z11974	macrophage mannose receptor precursor
Il4ra	M27960	interleukin 4 receptor, alpha
Rrm2	M14223	ribonucleotide reductase M2
Npn3	Z31362	
Col5a1	AB009993	collagen a1(V)
Cyba	M31775	
	AF010499	guanidinoacetate methyltransferase
Apbb1ip-pending	AF020313	proline-rich protein 48
Abca1	X75926	ABC transporter
Cmkar4	Z80112	CXCR-4
Cdk7	X74145	protein kinase
2310031E04Rik	AW230891	
Ifnar2	Y09864	soluble type I interferon receptor subunit
Tuba6	M13441	tubulin alpha 6
Fcgr1	M31314	Fc receptor, IgG, high affinity I
Ifi204	M74123	
Pfc	X12905	properdin (AA 5 - 441)
Scyb14	AW120786	
Capg	X54511	Myc basic motif homologue-1
Myo5a	X57377	myosin heavy chain

TABLE 1

beta 1	L48687	voltage-dependent Na+ channel beta-1 subunit
Myla	M19436	myosin light chain
2410045D21Rik	AI573601	
Msn	AI839417	
Sparc	X04017	secreted acidic cysteine rich glycoprotein
1300002F13Rik	AI853531	
8430417G17Rik	AI225296	
Ddah2	AF004106	dimethylarginine dimethylaminohydrolase 2
beta ig-h3	L19932	p68(beta ig-h3)
D5Wsu111e	AA790307	
Gstm3	J03953	
A12	L22977	X-linked lymphocyte-regulated 3b
Cebpb	M61007	alpha-1-acid glycoprotein
	AI841076	
AW549277	AI845902	
flp	M16238	fibrinogen-like protein
1810027D10Rik	AI504305	
Eln	AA919594	
Btg2	M64292	B-cell translocation gene 2, anti-proliferative
Col6a2	Z18272	collagen alpha 2 chain type VI
Peg3	AV353105	
Anxa2	M14044	calpactin I heavy chain
Cebpd	X61800	C /EBP delta
Apod	X82648	apolipoprotein D
Pnp	U35374	purine nucleoside phosphorylase
Ctsl	X06086	cathepsin L
Glk	AV217354	
Il1r2	X59769	type II interleukin-1 receptor
Cd48	X53526	BCM1 antigen
2900055D03Rik	AI839395	
1110032A03Rik	AI851206	
MRP14	M83219	intracellular calcium-binding protein
Fosb	X14897	FBJ osteosarcoma oncogene B
C33, Cd82, KAI1	D14883	C33/R2/IA4
Tnfrsf1b	X87128	p75 TNF receptor
0610011I04Rik	AI787183	

TABLE 1

Tubb2	M28739	
Pstpip2	Y18101	macrophage actin-associated-tyrosine-phosphorylated protein
Shc1	AI050321	
THBS2	L07803	thrombospondin 2
Actx	J04181	melanoma X-actin
Hp	M96827	haptoglobin
Hipk3	AF077660	homeodomain-interacting protein kinase 3
Fxyd5	U72680	ion channel homolog RIC
Bgn	X53928	biglycan (PGI)
Fbn-1	L29454	fibrillin
oxyR	L35599	Y-box binding protein
	AI839289	
Hspa2, HSP70A2	M20567	heat shock protein
Lbp	X99347	LPS-binding protein
C3ar1	U77461	anaphylatoxin C3a receptor
Col1a2	X58251	pro-alpha-2(I) collagen
Cldn5	U82758	lung-specific membrane protein
Pva	X59382	parvalbumin
Lcp2	U20159	SLP-76
Ampd3	D88994	AMP deaminase 3
Col1a1	U03419	alpha-1 type I procollagen
Peg3	AW120874	
Ier3	X67644	
Nfe2l1	AF015881	nuclear factor erythroid-related factor 1
Epcs21-pending	AI853172	
Madh1	U58992	mSmad1
Eif4ebp2	U75530	PHAS-II
Macs	M60474	myristoylated alanine-rich C-kinase substrate
Col6a1	X66405	collagen alpha1 type VI-precursor
	AI019679	
Fn1	M18194	
Krt1-10	V00830	
Grb10	AF022072	adapter protein
	X58196	H19 fetal liver mRNA
C76746	C76746	
Ensa	AJ005985	alpha-endosulfine

TABLE 1

helix-loop-helix protein Id2	AF077861	inhibitor of DNA binding 2
Prkar2a	J02935	
Ctsh	U06119	cathepsin H prepropeptide
2510015F01Rik	AW060556	
Txn	X77585	thioredoxin
Bmp1	AA518586	
Clast1	AB031386	Clast1
Ptx3	X83601	pentaxin related gene
Lxn	D88769	latexin
Cyba	AW046124	
Maged2	AI851574	
2310042E05Rik	AI839731	
Top1	X70956	topoisomerase I
Rnf13	AF037205	RING zinc finger protein
	AA189811	
1300002F13Rik	AW212475	
Sox4	AW124153	
AI413331	AA796989	
JNK2, Prkm9, p54aSAPK	AB005664	JNK2
Tctex1	M25825	t-complex testis expressed 1
Ly111, entactin-2	AB017202	entactin-2
D15Ertd781e	AI528219	
Serpinf1	AF036164	pigment epithelium-derived factor
MS1	L26479	elongation factor-1 alpha
	N28179	
Srst	X67863	simple repeat sequence-containing transcript
Col18a1	L22545	alpha 1(XVIII) collagen
Dnajb9	AW120711	
1200003O06Rik	AI315650	
AW558171	AW120868	
Gus-s	M19279	beta-glucuronidase structural
Snx2	AI842754	
Pftk1	AF033655	Pftaire-1
Ifi30	AI844520	
9130211I03Rik	AA711915	
fisp-12	M70642	FISP-12 protein

TABLE 1

Tgfb2	X57413	transforming growth factor-beta2 precurser
Pltp	U28960	plasma phospholipid transfer protein
Cd53	X97227	CD53 antigen
Ncam	X15052	neural cell adhesion molecule NCAM-180
Tnp1	X12521	transition protein 1 (during histone to protamine replacement)
S100a11	U41341	endothelial monocyte-activating polypeptide
Adm	U77630	adrenomedullin precursor
Tff1	Z21858	pS2m
	AI849721	
Ctsk	AJ006033	cathepsin K
Mapkapk2	X76850	MAP kinase-activated protein kinase 2
Cpo	D16333	coproporphyrinogen oxidase
1600017F22Rik	AV268207	
cyp C	M74227	cyclophilin C
Klkbp	X61597	kallikrein-binding protein
Plod3	AI840146	
3110004L20Rik	AW123347	
edr	AJ007909	erythroid differentiation regulator
2310038G18Rik	AI851313	
	AA002843	
6530405F15Rik	AI644072	
Rbp1	X60367	cellular retinol binding protein I
Nfil3	U83148	NFIL3/E4BP4 transcription factor
AI173274	AI642389	
Gzma	M13226	granzyme A
Myod1	M18779	myogenic differentiation 1
Lama4	U69176	laminin alpha 4 chain
Ig Vheavy-PCG-4	X82692	
Wsb1	AF033186	WSB-1
Tm7sf1	AI060729	
1110004C05Rik	AW125390	
Sap30-pending	AF075136	Sin3-associated protein
AU046135	AI842065	
R75394	AI852838	
Acta1	M12347	alpha-actin
Gltp-pending	AI842825	

TABLE 1

Fap	Y10007	fibroblast activation protein
Osmr	AB015978	oncostatin M receptor beta
AW122239	AW122239	
Numb	AV377244	
Dab2	U18869	p67; p96; p93
Actb	M12481	
Atp6n1	U13836	vacuolar adenosine triphosphatase subunit Ac116
1500001M20Rik	AV322862	
Bgn	AV166064	
Il6st	X62646	gp130
	AI593759	
6330407G11Rik	AV341723	
Gapd	M32599	glyceraldehyde-3-phosphate dehydrogenase
2310010N19Rik	AV335997	
CD106, VCAM-1, Vcam-1	M84487	vascular cell adhesion molecule-1
Capn6	AI747133	
Peg1/MEST	AF017994	Peg1/MEST protein
mptp	M80739	protein tyrosine phosphatase, non-receptor type 2
Evi2	M34896	ecotropic viral integration site 2
Laptm5	AV356071	
sprouty4	AB019280	sprouty-4
Eif1a	AI132207	
5830413E08Rik	AI849939	
Nucb2	AJ222586	precursor NEFA protein
sid478	AB025408	sid478p
Pik3r1	U50413	phosphoinositide 3-kinase p85alpha
Ier2	M59821	growth factor-inducible protein
1300003H02Rik	AW123556	
shrm	AI641895	
Abcc1a	AF022908	multidrug resistance protein
Arhc	X80638	p21RhoC
Mkrn1	AW125438	
hr	Z32675	hairless protein
AI428538	AW048730	
Tieg	AF064088	transcription factor GIF
Col15a1	AF011450	type XV collagen

TABLE 1

	AW046449	
Trt	AW122985	
COL9A1L, D6S228E	AB000636	collagen a1 XIX chain
alpha-1 gap junction	M63801	connexin 43
3110003A17Rik	AA833425	
D7Ertd304e	AI157475	
Grb2	U07617	Grb2 adaptor protein
Nramp	L13732	integral membrane protein
TXNRD1	AB027565	thioredoxin reductase 1
1810003P21Rik	AI844626	
2810417H13Rik	AI122538	
PLA2	M72394	phospholipid-binding protein
Mfap5-pending	AW121179	
Ptprc	M14343	protein tyrosine phosphatase, receptor type, C
Mx1	M21038	Mx1 protein
C80305	AI848825	
Ppicap	X67809	peptidylprolyl isomerase C-associated protein
4922501H04Rik	AI836718	
Ifi204	M31419	interferon-activatable protein
CMH2	L47600	cardiac troponin T
ST2L	D13695	ST2L protein precursor
Acinus-pending	AI839299	
Ifi204	M31419	interferon-activatable protein
Cstb	U59807	cystatin B
	D49733	lamin A
Rpl3	Y00225	J1 protein
Rgs2	U67187	G protein signaling regulator RGS2
Ankrd2	AJ011118	skeletal muscle and cardiac protein
Atp2a1	X67140	mouse fast skeletal muscle SR calcium ATPase
14-3-3 zeta	D83037	14-3-3 zeta
Eif4ebp1	U28656	PHAS-I
Tmsb10	AI852553	
TLR6	AB020808	TLR6
Apobec1	U22262	apolipoprotein B mRNA-editing component 1
2610318G08Rik	AA982595	
Islr	AB024538	ISLR

TABLE 1

Bcat2	AF031467	branched-chain amino acid aminotransferase
Krt2-4	X03491	keratin complex 2, basic, gene 4
Mch6, ICE-LAP6, Caspase-9	AB019600	caspase9
Lgl	M34597	immunoglobulin lambda-chain
1110034C02Rik	AI837104	
AI415285	AW049806	
Dlixin, Dlxin1, Dlxin-1,	AB029448	Dlxin-1
Ctsc	U74683	dipeptidyl peptidase I precursor
Mknk2	AI845732	
2810411G23Rik	AI854343	
S100a13	X99921	S100 calcium-binding protein A13
Dscr1	AI846152	
ADFP	M93275	adipose differentiation related protein
Hif1a	Y09085	hypoxia-inducible factor one alpha
Slc16a2	AF045692	X-linked PEST-containing transporter
AA575098	AA575098	
Hif1a	AF003695	hypoxia-inducible factor 1 alpha
EFP, Zfp147	D63902	estrogen-responsive finger protein
Rcal	D13003	reticulocalbin
Ogn	AA647799	
3110046C13Rik	AI172819	
AU043077	AA212964	
AI596360	AI596360	
1810049E02Rik	AA763937	
	X05546	
1110064N10Rik	AW124599	
1110036C17Rik	AW123191	
grg	L12140	amino-terminal enhancer of split
1200007D18Rik	AA815795	
1200012G08Rik	AA880988	
murine CD63	D16432	murine homologue of CD63/ME491
Vps16	AI847040	
4632435C11Rik	AF017639	carboxypeptidase X2
Col6a1	AV010209	
Krt2-16	AV085755	
GTPCH, GTP-CH	L09737	GTP cyclohydrolase I

TABLE 1

C77137	C77137	
AA589446	AI849075	
kr, Krml, MafB	L36435	basic domain/leucine zipper transcription factor
Xin	AF051945	Xin
Dnajc3	U28423	p58
Slpi	AV090497	
Surf5	AV264321	
1190002H23Rik	AI854358	
Cma1, Mcp-5, MMCP-5	M68898	chymase 1
Dnajc3	U28423	p58
1110025H08Rik	AV360058	
0610008L05Rik	AV380793	
D7Wsu105e	AA388099	
	AF073881	myotubularin homologous protein 3
Apaf1	AF064071	apoptotic protease activating factor 1
	AW125241	
P3, DXS253Eh, DXSmhG28	J04761	
Jup	M90365	plakoglobin
p50, WP34, pp52, Lsp-1	D49691	p50b
TMEFF2	AB017270	transmembrane protein with EGF-like and two follistatin-like domains 2
AI853222	AW124544	
AI132321	AW123773	
Adcy7	U12919	adenylyl cyclase type VII
AA407055	AI550305	
	AI837786	
Ednra	AI180687	
Dtx1	U38252	FX-induced thymoma transcript
Aldo1	Y00516	aldolase 1, A isoform
Pros1	L27439	protein S
Diap1	U96963	p140mDia
AI181838	AV316991	
Mmp14	AF022432	matrix metalloproteinase-14
	AI847033	
A1b	U23778	A1-b protein
Usf2	X77602	transcription factor
D730045A05Rik	U69488	viral envelope like protein

TABLE 1

C76222	AI846773	
Fosl2	X83971	fos-related antigen-2
Pim1	AA764261	
Midn-pending	AW124785	
1700017B05Rik	AW049360	
Sod3	U38261	extracellular superoxide dismutase
Gnb1	U29055	G protein beta 36 subunit
Psma5	AW048997	
Peg3	AF038939	zinc finger protein
AU021460	AI131895	
Igfbp3	AI842277	
2310021G01Rik	AI606257	
Akap12	AB020886	SSeCKS
CDK2	AJ223733	cyclin-dependent kinase 2
Ap3s2	U91933	AP-3 complex sigma3B subunit
Uck2-pending	AI850362	
Fbln1	X70853	BM-90/fibulin
Serpinh1	X60676	heat shock protein
Zfp106	AF060245	zinc finger protein 106
MD1, MD-1	AB007599	lymphocyte antigen 86
1200017E04Rik	AW048159	
G6, Clcp	AF109905	Hsc70t; smRNP; G7A; NG23; MutS homolog; CLCP; NG24; NG25; NG26
Ppp4c	AF088911	protein phosphatase X
Arih2	AJ130975	Ariadne-2 protein (ARI2)
Rab7-ps1	Y13361	
3230402M22Rik	AW122364	
Atp6a2	AW123765	
Col6a3	AF064749	type VI collagen alpha 3 subunit
B220, CD45, Cd45, Ly-5, T200, CD45R, Lyt-4	M23158	protein tyrosine phosphatase, receptor type, C
	AA397054	
MSGP-2	D14077	sulfated glycoprotein-2
	AA710439	
AI482343	AW123850	
Cdkn1c	U22399	p57KIP2
C1r	AI132585	
epithelin	D16195	acrogranin precursor

TABLE 1

Lipo 1	M69260	lipocortin I
C10	M58004	small inducible cytokine A6
Tnfrsf1a	X57796	55kDa tumor necrosis factor receptor
EGFR	L06864	epidermal growth factor receptor
Lum	AF013262	lumican
Cpt1a	AF017175	carnitine palmitoyltransferase I
Ly6	X04653	lymphocyte antigen 6 complex
Pdk4	AJ001418	pyruvate dehydrogenase kinase-like protein
Slfn2	AF099973	schlafen2
	AB022316	semaphorin W
Col9a3	AW212495	
Gadd45g	AF055638	growth arrest and DNA-damage-inducible 45 gamma
HB-EGF	L07264	heparin-binding EGF-like growth factor precursor
Lor	U09189	loricrin
tPA, t-PA	J03520	plasminogen activator, tissue
Ppp1r5	U89924	protein phosphatase 1 binding protein PTG
Hsp70-3	M12571	68 kDa heat shock protein
A1d	U23781	A1-d protein
Npn1	Z31360	
Psmd4	AF013099	multiubiquitin-chain-binding protein
Fkbp5	U16959	FKBP51
Ptk9l	Y17808	A6 related protein
Igfbp4	X76066	insulin-like growth factor binding protein 4
Ryr3	X83934	ryanodine receptor type 3
1110027O12Rik	AW212271	
LOC55989	AF053232	SIK similar protein
Mglap	D00613	MGP precursor
4921531N22Rik	AI196645	
	AI841493	
Nfkbia	U57524	I kappa B alpha
Capn3	X92523	calpain
Car2	M25944	
Ces3	AW226939	
Grim19-pending	AI854527	
Cyp2e1	X01026	
adrenodoxin	L29123	iron-sulfur protein

TABLE 1

Ckmt2	AV250974	
D16Bwg1543e	AI573367	
Lipe	U69543	hormone-sensitive lipase
Acrp30	U49915	adipoQ
Cycs	X01756	cytochrome c
	AI118905	
myosin light chain 2	M91602	myosin light chain 2
J chain	M90766	joining chain
Aqp4	U88623	aquaporin-4
Retn	AA718169	
Temt	M88694	thioether S-methyltransferase
Mrps7	AI848784	
Igk-V28	M18237	
H2afy	AA646966	
TIMP-3	U26437	tissue inhibitor of metalloproteinases-3
AW047450	AW047450	
Clcn3	AF029347	chloride channel protein 3
Fmo1	D16215	flavin-containing monooxygenase
2900062L11Rik	AI839718	
	AI852124	
mld, shi, Hmbpr	M11533	myelin basic protein
Cdo1	AI854020	
Amd2	Z23077	S-adenosylmethionine decarboxylase
	AW212131	
Stat1	U06924	Stat1
Rasd1	AF009246	ras-related protein
Aqp4	U48398	mercurial-insensitive water channel 2
MLP, CRP3, MMLP	D88791	muscle LIM protein
Cd1d1	M63695	CD1.1
Mapbpip-pending	AI844560	
Adsl	AA606587	
Akl3l-pending	AI854743	
Fasn	X13135	fatty acid synthase (838 AA)
AA959601	AW125299	
Gstz1	AW060750	
Thrsp	X95279	Spot14

TABLE 1

Ldh2	X51905	lactate dehydrogenase 2, B chain
AI848390	AW045204	
Amd2	Z23077	S-adenosylmethionine decarboxylase
Enpp2	AW122933	
Apobec2	AW124988	
Myhcb	AJ223362	slow myosin heavy chain-beta
2310032D16Rik	AW125284	
1110007M04Rik	AA693236	
5730469M10Rik	AI850090	
Gdm1	D50430	glycerol-3-phosphate dehydrogenase
Myh11	D85923	myosin
	AW047232	
0610042C05Rik	AW048828	
	AW047643	
2610100P18Rik	AW123099	
AAAT, ASCT2	L42115	insulin-activated amino acid transporter
	AA733664	
1110004O20Rik	AW060921	
	AI197161	
AW060987	AI841606	
Pfkfb1	X98848	6-phosphofructo-2-kinase /fructose-2,6-bisphosphatase
Ms4a2	AA797989	
Slc25a15	AA986782	
Iigp-pending	AA914345	
C80633	AI853240	
Tncc	M29793	troponin C, cardiac/slow skeletal
2610042L04Rik	AI853444	
0610011L04Rik	AI849271	
	AI851321	
AA420417	AW123788	
2310061N23Rik	AI158810	
Bet1	AF007552	Bet1p homolog
Gdc1	M25558	glycerolphosphate dehydrogenase 1, cytoplasmic adult
MLC1s, MLC1v	X12972	
Tpm5	U04541	alpha-tropomyosin slow
Mrps25	C77227	

Table 2. Up-regulated genes following femoral artery ligation

Gene Name	Accession number	Femoral artery ligation					Sham				
		6 hour	1 day	3 day	7 day	14 days	6 hour	1 day	3 day	7 day	14 day
Angiogenesis											
Cyr61	M32470	3.10	2.04	3.03	3.01	1.66	1.28	2.73	2.89	2.51	
Fgfrp	U04204		2.35	3.49	2.88	2.06			2.25	1.80	
Fin14	U42386	1.26	2.47	0.91	0.91	1.11	1.11	0.96	0.77	0.87	0.94
Hdgf	D63707	2.97	2.01	2.67	1.94	1.41		2.09			
IP10 (scyb10)	M33266		2.69	2.89	4.10	2.57		3.17	3.16	1.64	
MIG (scyb9)	M34815		1.25	0.88	2.77	0.81			0.83	0.72	
MCP1 (scya2)	M19681	9.94	28.8	12.5	3.61	2.61	7.67	10.82	6.11	1.90	
PlGF	X80171		3.46	1.76							1.94
TGFβ1	AJ009862				15.7	21.7					
Cell growth and survival											
Btg1	Z16410	0.74	2.11	1.2	1.36	1.05	0.62	0.92	1.40	0.92	0.35
Casp3	U54803		1.00	1.33	2.22	1.63				0.719	
Ccnb1-rs1	X64713			53.8	19.5				31.3		
Ccnt1	AF095640	3.25	1.70	2.45	1.78	1.77				1.80	2.63
Cdc2a	M38724			4.12	2.61				1.97		
Cdkn1a (p21)	U09507		2.86					0.98			
	AW048937	3.03	5.38	2.36	3.45	2.48	2.78	2.30	1.72	1.72	
Cdkn1c	U22399	1.15	1.09	0.73	5.60	2.84	0.90	0.89	0.71	1.74	1.0
Dck	X77731			1.90	2.75	2.05				1.17	

Table 2 (cont'd.)

Gadd45a	U00937	1.36	12.1	2.11	1.51	1.56	2.38	2.62	2.83	1.21	0.42
Gadd45b	AV138783	5.13	0.93		0.89	0.65	1.79		0.71	1.13	1.10
Gas2	M21828		1.28	1.09	2.71	2.29				1.57	
Gas5	AI849615		2.36	1.08	1.0					0.85	
	X59728				12.8						
Grb10	AF02207	1.93	0.76	1.18	3.19	2.85	2.46	1.51	0.91	0.75	
	U18996				5.59						
Hmox1	X56824	3.26	8.52	5.21	1.58	1.71	5.36	2.85	3.29	1.24	0.96
Hnrpu	AF073992				2.61			2.978			
HSP70A2	M20567		3.03	1.85	2.11		2.76	1.49	2.69	1.50	
HSP70-3	M12571	2.11	5.36	0.67	0.95	0.79	1.01	1.07	0.69	0.75	0.62
Hsp86-1	AV358673				3.92						
Lcn2	X81627	2.56	60.6	10.2	0.97	1.02	5.46	8.64	5.57		
Mki67	X82786			2.81	2.62	1.70			1.34		
Mt1	V00835	8.14	40.8	20.4	1.94	0.76	17.9	11.4	17.4	0.79	
Mt2	K02236	19.0	38.3	30.2	3.6	1.80	36.3	29.9	30.6	1.15	0.97
Mts1	M36579	0.73	3.50	7.10	3.79	3.75	1.18	4.83	3.46	3.30	1.19
Np95	D87908			3.98							
Perp-pending	AI854029		4.87		1.89			1.46	2.14	0.77	
Pftk1	AF033655		1.35	1.32	2.42	2.91		2.12	1.57	2.0	
POLA1	D13543			2.16	1.41	1.78		1.70		1.30	
Rex3	AF051347			2.98	7.11	2.85	2.55	2.39	1.45	2.72	
Sepp1	AF021345	4.48		2.25	3.85	2.88		3.09		2.77	
SGP-1	AF037437	42.1		29.4	27.4	47.6	59.6				
Tdag	U44088	2.72	5.73	3.28	3.89	1.93	2.79	2.36	1.43	1.96	

Table 2 (cont'd.)

Tiap	AB013819			3.04	2.75	1.7			2.84	1.16	
Cell shape and motility											
A1b	U23778		1.92	2.09	1.66	2.35		1.47	1.08	1.10	
Ap1g1	X54424	2.03	1.39	1.12		1.10				0.45	
Ap3s2	U91933			2.35	1.89	2.04		1.51	1.28	1.29	
CMH2	L47600	1.26	0.82	0.89	8.82	3.44		0.69	1.30	1.45	1.36
Crp2	D88792			3.69	8.74	5.13		2.80	3.32	4.38	
Cttn	U03184		3.23	2.13	3.53	3.64	3.86			2.35	
Dmdl, G-utrophin	Y12229	1.75	0.90	1.034	1.13	2.18	1.33	1.38	1.0	1.28	1.05
Fbln2	AV321999					3.80	8.65				
Jup	M90365	1.83	2.40	1.56	1.63	1.84	1.89	0.81	0.86	1.58	2.05
Lmnb1	M35253	3.52	2.23	3.62	2.41				1.08	1.68	
Mlp	X61399	1.91	2.98	2.63	2.80	2.47		2.03	1.72	1.23	1.91
Myhse	M74753			1.79	54.0	9.46			2.99	3.33	
Myh8	M12289		1.61	1.15	24.1	11.1	2.29	1.49	1.47	3.54	2.64
Myla	M19436		0.81	1.43	32.6	14.32		1.08	2.87	6.76	1.62
pgM	D45889		5.69	3.13	3.26	3.58		3.35	2.86	2.69	
Tmsb10	AI852553	0.39	0.96	1.96	4.15	1.71	0.36	0.92	2.00	1.34	0.71
Tubb2	M28739	0.96	1.54	3.39	3.91	2.05	0.72	1.63	2.14	1.52	1.02
Tubb5	X04663	0.86	2.14	3.53	2.72	2.12	0.82	1.77	2.22	1.48	0.71
Cytokines and Inflammation											
Anxa1	AV003419	0.77	1.37	2.62	2.36	2.28	0.32	1.73	2.19	2.32	0.71
Anxa2	M14044	0.97	2.84	3.11	1.90	2.43	0.80	2.62	1.94	1.86	1.04
BAP, Bap3	AC002397				2.83	1.936					
		2.38	1.47		1.68	1.61				1.17	

Table 2 (cont'd.)

BV13S1	AE000664		11.9								
CD14	X13333	2.33	8.23	2.53	2.56	2.25	2.04	3.29	1.91	1.38	
CD68	X68273		2.47	5.05	4.41	3.23		2.41	3.06	1.34	
CD180	D37797	1.57		1.53	1.61	2.46				1.42	
Cmkbr1 (CCR-1)	U29678		10.6	5.72		3.33		7.39	3.46		
Cmkar4 (CXCR-4)	Z80112		1.07	2.54	3.72	2.52			2.30		
Cmkbr5 (CCR-5)	AV370035		7.35	12.63	4.61	5.16		6.86	7.12	2.52	
Cmkbr5	X94151		12.1	29.0		13.2		17.1			
Csf3r	M58288		16.3								
Csf1r	X06368	0.74	1.14	2.99	4.30	2.97	0.60	2.27	2.01	2.60	1.26
Ctla2a	X15591	1.02	4.13	1.86	0.83	0.77	1.06	1.28	1.49	0.75	
Ela-1, PC-TsF	M27347		2.12	2.36					1.94		
Emp1	X98471	1.90	3.93	2.87	3.17	2.78	2.37	2.52	2.15	2.21	0.95
Fcgr1	M31314	0.85	2.91	3.67	2.45	1.75	1.20	2.68	1.78	0.94	1.00
Fcgr2b	M31312		2.77	4.80	2.53	2.54		2.79	4.89	1.56	
Gp49	M65027	2.29	14.9	6.42	5.23	4.89	1.69	4.98	3.28	2.22	0.91
Gp49b	U05265	2.53	9.81	4.25	3.78	3.76	3.08	5.55	2.71	1.60	
IL1β	M15131		240	49.9				107	50.8		
IL1r2	X59769	3.53	7.3	1.14	0.93	1.39	3.47	2.64	1.79	1.01	0.99
	AV223216		9.90	2.78				3.03			
IL1-rn	L32838		31.0	10.7		14.8		10.9			
IL4ra	M27960	1.79	5.48	4.57	1.47	1.03	2.403	2.163	3.29		
IL6	X54542	255	211	161			114	150	161		
IL17R	U31993	3.49	4.58	3.50	1.45	1.20	3.34	2.21	2.42	1.35	1.50
Iigp-pending	AJ007971	1.12	1.32	1.0	3.79	1.01	1.18	0.74	0.92	0.99	0.68
Ifi30	AI844520		1.40	4.50	2.54	1.94		1.40	4.33	0.96	

Table 2 (cont'd.)

Lcp2	U20159		2.57	3.69	2.16	1.13	1.02	1.63	3.63	1.07	
Lgals3	X16834	0.60	5.38	7.98	7.56	9.16	1.02	3.40	4.99	2.52	1.03
Ly68	AF081789	0.77	0.99	2.03	1.88	1.39	0.63	1.22	0.76	1.11	0.58
Ly57	AF068182			1.63	2.90	1.38			2.28		
Lzm	M21050	0.53	1.52	3.28	3.59	3.99	0.23	2.98	3.13	2.36	0.98
Lipo1	M69260	0.82	1.44	1.98	2.14	2.69	0.69	1.84	1.90	1.84	0.81
Lta	M17015			1.51	3.05				2.07		
Mincle	AB024717		7.59								
Mpcl	AF061272		79.4	21.2		16.0		24.6	10.3		
MRP8	M83218	2.01	10.2	2.74	0.53	1.04	1.12	3.49	4.30	0.14	
MRP14	M83219	2.06	9.16	1.9		0.72	1.44	3.46	2.99		
Pim1	AA764261	2.0	3.20	1.58	1.65	1.85	1.87	1.39	1.55	1.73	2.02
Ptn	D90225				8.93	8.89					
Ptx3	X83601	4.1	7.93	1.32	0.97	0.71	3.12	1.98	1.32	1.38	
Psme3	AB007139	1.99		2.24	2.18	1.751					
Saa2	U60438		10.5								
Saa3	X03505		23.9	12.0	1.97	7.69	3.20	15.2	43.7	1.67	2.81
SCGF	AB009245				2.61	1.78				1.22	
Scya3 (MIP-1α)	J04491		6.75								
Scya7 (MCP3)	X70058	4.96	34.5	15.24	4.33	2.36	5.49	18.3	7.55	2.12	0.44
Scya9 (MIP-1 γ)	U49513	1.09	9.96	7.76	1.76	3.25	2.23	11.38	3.87	2.43	1.04
Scyb2 (MIP 2)	X53798	52.3	394	24.9	6.43	31.8	28.2	112	24.1		
Scyb5 (ENA78)	U27267	9.57	266	36.6		9.10	27.43	60.7	34.3		
Scyb14	AW120786	2.11	5.34	2.83	1.30	1.14	1.70	2.35	1.73	1.14	1.03

Table 2 (cont'd.)

Selpl	X91144		1.7	2.35	1.02	1.65			1.13	0.79		
Slfn3	AF099974		10.5	1.97				5.2	2.39			
Slfn4	AF099977	1.95	23.5	4.99	1.30	2.26	4.42	19.8	2.45	0.83	0.57	
Slpi	AF002719		7.03	6.49	0.45	1.83		2.83	7.79		2.53	
Tnfrsf1b	X87128	0.94	2.04	2.25	2.17	2.24	1.56	1.42	1.68	1.36	0.98	
Tnfip6	U83903	2.44	2.85	2.16	3.31	1.79	1.30	4.66	3.74	1.50		
Wsb1	AF033186	1.24	1.85	2.04	2.66	2.47	0.96	1.37	1.89	1.32	1.02	
Extracellular matrix												
Anxa4	U72941	0.55	1.53	4.07	2.48	2.28	1.07	2.54	3.21	1.67	0.93	
Anx5	D63423	0.88	0.94	1.73	1.86	2.08	0.72	1.38	1.26	1.68	1.00	
Bgn	X53928	0.99	1.12	2.71	6.92	4.70	0.78	1.59	1.71	3.19	1.09	
Bmp1	AA518586	2.38	1.57	1.87	2.38	3.11				1.57	1.87	
C1qa	X58861	0.89	0.68	2.98	4.13	3.68	0.88	1.29	2.57	2.07	1.35	
C1qb	M22531	1.36	1.05	5.02	6.07	4.36	1.16	1.67	3.92	2.38	1.66	
C1qc	X66295	1.24	1.01	3.26	4.51	3.39	0.94	1.50	2.81	2.09	1.04	
Cathepsin K	AJ006033	1.50	1.21	1.22	2.51	3.92		0.93	1.30	2.29	1.49	
Cathepsin S	AJ223208	0.25	1.47	5.17	5.38	4.35	0.32	2.35	3.24	1.93	0.52	
Cathepsin Z	AJ242663	0.49	1.07	1.79	2.55	1.67	0.43	1.05	1.98	1.19	0.63	
CD106 (VCAM-1)	M84487		0.31	2.23	2.76	1.30		0.81	3.91	1.08		
Ceacam2	AF101164	2.41			1.68				1.77	1.34	2.33	
Cdh2	M31131		0.79	1.94	3.68	2.66			1.79	1.73		
Col1a1	U03419	1.78	0.90	2.67	6.90	8.51	1.09	1.10	1.88	4.92	1.98	
Col1a2	X58251	0.95	1.10	2.10	6.35	7.35	0.90	1.26	1.73	3.97	1.14	
Col3a1	AA655199	1.49	1.20	5.30	9.94	12.8	0.69	2.39	5.28	7.42	1.93	

Table 2 (cont'd.)

Col5a1	AB009993	2.05		2.46	6.19	7.10			1.42	3.14	1.78
Col6a2	Z18272	1.80	0.84	2.10	5.63	5.58	1.24	0.87	0.95	3.23	1.57
Col18a	L22542	1.60	0.84	1.68	3.49	2.93		1.35	1.44	2.10	
Col8a1	X66976		1.21		4.99	2.96				1.84	
Col15a1	AV112006					2.01				0.97	
COLQ	AF021231	2.65									
CPX-1	AF07773			18.0	19.9	15.0					
Eln	U08210	3.5		1.62	3.56	6.28			1.24	2.21	3.26
Fmod	X94998	7.05			12.7	7.01		2.38		4.86	
Has2	U52524				10.8	6.19				6.27	
Lama4	U69176		0.82	1.48	2.24	2.32		1.47	1.31	2.01	
Lgmn	AJ000990	1.17	2.99	5.58	6.49	4.40	0.88	2.98	5.20	2.07	1.22
Lum	AF013262	0.45	0.44	0.95	1.80	2.6	0.47	0.72	1.49	2.10	0.69
Ly111	AB01720			1.66	2.88	2.45		0.75	1.45	2.06	
Ly-24, Pgp-1	X66084		2.34								
Magp	L23769				9.56	10.0				4.28	
Mglap	D00613	0.81	1.52	1.24	4.21	2.65	0.62	1.75	1.26	1.65	0.61
MMP3	X66402		8.30	7.86	3.30	5.08		9.40	14.3	3.67	
MMP12	M82831			5.44	17.8	58.9				6.21	2.06
	M82831			11.86	18.9	83		1.72		8.9	3.95
MMP13	X66473			1.78	11.6	8.15					
MMP14	AF022432	1.05	0.84	1.35	2.55	3.15	1.14	1.26	1.16	1.85	1.21

Table 2 (cont'd.)

MT2-MMP	D86332				2.87	3.73				2.77	
OSF-2	D13664	0.94	1.22	3.12	25.9	18.2	0.52	1.35	2.89	10.7	1.26
PAI-1	M33960	3.34	5.57	2.13	2.95	1.33	2.7	1.36	1.92	1.25	0.71
Plaur	X62700		17.9					6.17			
Prg	M34603		3.08	1.51			1.21	1.54	1.90	0.92	
Rrg	D10837		8.22	8.96	11.7	6.72		3.87	6.23	3.35	
Spp1	X13986		11.1	32.7	14.6	19.5		10.8	9.45	2.81	
Sparc	X04017	1.72	1.08	2.05	3.99	5.16	0.67	1.05	1.74	2.65	1.06
Serpin	X60676	1.22	1.28	1.76	3.42	3.44	0.87	1.55	1.26	1.84	1.00
Serpinf1	AF036164	1.58	0.72	1.58	3.12	2.46	1.04	1.87	1.16	2.40	1.25
Tfpi	AF00483		2.03	3.78	3.91	2.81			2.90	2.32	
Tgfb1i4	X62940	1.06	3.38	0.94	1.98	1.34	0.92	1.22	1.25	1.63	0.79
Tgfb1	AJ009862				14.7	21.7					
Thbs1	M62470	2.82	17.9	4.79	7.96	3.68	1.72	10.9	4.40	2.29	0.66
TIMP	V00755	2.17	12.6	9.18	5.90	5.96	3.16	7.60	6.51	3.20	2.09
Tnc	X56304	1.65	3.35	2.99	19.9	8.78		1.64	4.36	3.82	
	AV230686			11.7	50.2	35.1			15.6	16.9	
TSC-36	M91380	1.77	1.48	2.45	3.99	4.58	1.58	1.57	2.00	2.95	2.13
Metabolism											
ABCA1	X75926			2.46	3.23	2.57		1.67	1.54	2.66	
Akr1c1	D45850		0.91			2.61					
Aldh1a3	AW050387		2.7	1.334	1.11		2.29		1.04	2.68	
Amy2	X02578		14.7	2.06					1.80		2.61
	X02578		252								

Table 2 (cont'd.)

Anpep	U77083			2.12	3.89	3.69				2.05	
Aoah	AF01817			2.11	3.84	2.75					
Apoe	D00466	0.67	0.83	2.58	5.90	4.17	0.61	0.94	2.81	2.79	1.26
Arg1	U51805		471	233		53.6		278	27.5		
Arg2	AF032466		2.37								
Atel	AF079097					2.084				2.158	
B3galt3	AF029792			2.28	2.0	3.0			2.52	2.09	
Car4	U37091		2.82								
Cel	U37386		2.58								
Cyba	M31775	0.68	2.04	2.29	3.07	3.19	0.67	1.67	2.03	1.37	0.73
Cyp3a16	D26137					65.3					
Cyp3a11	X60452					55.1					
Cyp3a25-pending	Y11995					16.6					
Cyp1b1	X78445		3.03	3.22	2.51	2.01		2.36	3.29		
CYP4A10	AB018421		6.41		7.62	10.13		8.48			19.1
Dda	AF071068			10.2	11.4	11.3			11.1		
Ddc	AF071068			10.3	11.4	11.1			11.1		
Dhcr7	AF057368			2.20							

Table 2 (cont'd.)

Es31	L11333					3.74					
Ftl	L39879	0.84	2.40	2.42	2.25	2.47	0.89	2.20	1.52	1.61	0.80
Fabp1	Y14660					44.0					
Gapd	AV008547	3.96	4.38	3.45	1.88				2.45		4.03
GCS	D89886		2.58	3.05	2.17	2.11	3.06				
Glns	U09114	0.82	3.26	0.58	0.84		1.53	1.07	1.46	1.11	0.55
Gm2a	U09816				1.45	2.07				1.63	
Gpx5	AV381732				2.09	1.17					
Gus-s	M19279	0.76	1.02	3.35	3.56	2.49	0.90	1.56	1.92	1.92	0.77
Hdc	X57437		37.7						12.0		
Hpgd	U44389				3.43	3.40			1.70	1.52	
Hsd17b3	U66827			2.52							
Lip1	Z31689			2.69	3.88	4.75					
Dihydrofolate reductase	J00388			5.72							
Lrp1	X67469	0.92	1.51	1.70	1.78	2.26	0.90	1.38	1.45	1.60	1.11
Man1a	U04299	2.31	1.76	3.01	1.68	2.27	1.60	1.11	3.46	2.29	
Man2b1	U87240			2.22	2.28	2.27			1.99	1.88	
Msr1	M59446		3.22	4.00	1.81	2.36		2.45	2.89		
Myeloperoxidase	X15378	3.82			2.55						
Pah	X51942		7.81			28.1					
Pcbd	AW04659				4.32	2.25					
Pdk4	AJ001418	1.82	4.06	0.83	0.61	1.07	2.49	1.97	0.94	1.14	0.81
Pitpnb	AI747899	3.87	1.83	2.50	3.89	2.17	5.33	1.84	1.61	1.24	
PLA2	M72394		1.38	1.94	1.96	2.27		2.55	1.43	1.63	
Pld1	U87868			2.01		2.94					

Table 2 (cont'd.)

Sat	L10244	2.22	4.59	2.18	1.97	1.54	2.52	1.50	2.22	1.79	
Slc2a1	M22998		3.86								
Tgif	X89749	1.38	9.19	3.13	2.68	1.24	1.67	2.38	3.57	1.83	0.95
Try2	X04574		32.4								
Ugt1a6	U16818		2.24	3.12	2.32	3.14		1.52	2.72	1.77	
Ugt2b5	X06358					6.54					
Uox	M27695					10.6					
Xdh	X75129	0.77	2.68	1.35	1.08	1.17	1.06	1.14	1.0	1.25	0.76
Signaling											
Activin	X69620		21.2	3.54							
Adam8 (CD156)	X13335		60.0	40.0	18.7			25.0			
Adcy7	U12919			1.78	2.36	2.01			1.89	1.86	
Akap12	AB020886	2.25	2.88	1.0	0.89	0.81	1.93		0.84	1.29	
Angptl4	AA797604	3.12	4.71	3.35	1.75	1.99	4.36		3.71	1.82	
Aogen	AF045887		2.16								
Bit (CD172a)	D85785			12.4	13.2	13.6					
	AB018194		1.78	4.85	7.03	4.74		2.93	2.89	2.35	
Bmk, Hck-1	J03023		2.64	3.57	2.74			2.44	2.51	0.82	
Btg2	M64292	3.33	3.71	1.35	1.84	1.01	2.81	1.69	2	1.80	

Table 2 (cont'd.)

C3ar1	U77461	0.34	1.99	5:21	4.36	2.87	0.59	2.20	1.70	1.78	0.75
C5aR	S46665	2.03	2.40	3.15	2.13	2.05	2.30	2.07	1.29	1.61	1.40
Calb3	AF028071				2.59	1.79					
Capn6	Y12582				19.2	8.53				3.97	
	AI747133	1.51	.87	1.31	5.69	3.66	1.26		1.09	2.01	1.8
CD116, GM-CSFR	M85078		6.81	6.27	8.47	9.24		4.51			
Chrna1	M17640			2.19	6.24	1.89			2.67	1.53	
Chrnb	M14537	0.6	0.95	1.44	2.07	1.65	0.66	0.77	1.38	1.25	0.46
Chrng	AV248455		6.17		24.6	7.32			8.97		
Cot	D13759		16.7								
Dab2	U18869		1.56	4.79	1.30	1.89		3.03	2.76	1.56	
Dok2	AF059583			3.34				3.20		2.66	
E3	U29539			2.68	2.73	2.82		1.18	1.59		
Ect2	L11316			3.58	2.71				3.13		
Ednra	AI180687	3.41	2.97	1.70	0.98	1.41	1.99	1.45	1.47	1.24	1.63
Egfr	AW049716			5.13							
ELAM-1	M80778			7.42				10.1			
Emr1	X93328			5.10	6.61	2.71		2.62	4.34		
F2rl1	AW046032	1.45		1.54	2.69	2.58		1.59	1.32	0.91	
Fap	Y10007	0.72		0.81	2.79	2.13	0.52	0.82	1.28	2.06	0.97
Fau	X65922				2.44	1.32					
Fkbp5	U16959	1.19	3.80	1.32	.53	.38	2.51	1.83	1.30	.76	
Fkbp10	L07063	2.25	1.76	2.43	3.69	3.53		1.80	2.03	2.05	
FPR	L22181		24.8								
Fpr-rs2	AF071180		18.4	4.75				8.30	5.03		
Gab1	AJ250669	3.03	2.21	2.51	2.16	2.58				2.95	

Table 2 (cont'd.)

gag-related peptide	X05546		1.52	2.48	1.98	2.27		0.96		1.39	
Gbp2	AJ007970		2.40	1.05	1.86	0.82			0.99	0.93	
Gnai2	AI841629			3.42	2.36						
Gna12	M63659			2.80	4.03	3.38					
Gngt2	AI882325			4.7	2.85						
Grb2	U07617	1.96	1.37	2.03	1.56	2.12	1.63	0.96	0.82	1.30	1.75
Gpcr25	U39827			3.63	5.36				4.47		
iba1	D86382			3.08	4.73						
Igf2	X71922				7.88	4.04				2.29	
Iigp-pending	AA914345	0.49	1.08	0.46	2.91	0.44	0.71	0.58	0.48	0.48	0.68
Impdh1	U00978				4.01						
Itga4	X53176	12.2		11.2	4.34						22.7
Itgax	AI035495				3.81	3.54					
Itgav	U14135	2.28	0.72			1.58					
Itgb2 (CD11b)	M31039		3.4	4.30	2.67	3.89		3.30	2.58	1.26	
Klkbp	X61597	1.41	3.53	1.58	0.63	1.44	2.19	1.18	2.86	1.09	1.06
Lerepo1-pending	AW049031	0.88	3.59	1.06	1.19	0.89	1.49	1.21	1.05	1.19	0.49
Macs	M60474	0.70	1.39	1.46	3.53	2.67	0.66	1.43	1.41	2.89	0.92
Map3k8	AV341985		2.16								
Mknk2	Y11092	3.86	2.55	4.20	2.41	2.18	3.36	1.84	1.62	1.44	3.07
Ncam	X15052	2.08	1.27	1.99	4.39	2.90	1.82	1.60	1.68	1.79	

Table 2 (cont'd.)

Nck1	AF084183				2.15				2.00		
NLRR-1	D45913	0.55	0.45	1.20	2.80	0.99	0.58	0.99	1.46	1.12	0.72
Nodal	X70514	4.58									
P50 (LSP1, pp52)	D49691		1.32	1.97	2.63	2.73		1.37	1.92	1.88	
Pi4k2-pending	AW121695		2.51		1.08	0.97			0.90	0.76	
Pik3r2	Y13569					2.28				2.61	
Pira3	U96684		4.40	2.98	2.35	3.33		2.02	2.18	1.43	
Pld3	AF02612			2.10	3.56	2.23					
Plk-ps1	U73170			4.18	3.18	2.74	4.14	3.73		4.68	
Ptgerep2	AB007696		3.26	4.47							
Ptpn12	X63440		2.67								
Rbp1	X60367			1.56	3.08	2.45		1.47	1.26	2.33	
Rcal (reticulocalbin)	D13003	0.56	1.18	1.70	2.36	2.06	0.57	1.23	1.79	1.96	0.48
Rrad	AF084466	10.4	17.5	6.89	5.17	3.39	26.3	9.42	10.3	3.32	1.36
S100a10	M16465	1.00	2.16	2.80	1.64	2.08	0.83	2.15	2.10	2.09	0.98
Sfrp2	U88567	1.08	0.70	4.92	2.81	6.04	0.90	1.06	1.99	9.84	2.61
Shc1	AI050321	1.25	1.74	2.02	2.38	2.80	1.07	1.77	1.40	2.01	0.94
Sphk1	AF06874		44.8								
Spi2-rs1	X69832		53.6	21.8			11.8	14.5	19.2		8.63

Table 2 (cont'd.)

Spi1-1	M75721				11.9						
Spi1-2	M25529		7.45		26.0						
Spi1-3	M75720		2.53		6.91						2.18
Spi1-4	M75718	3.15	4.69	1.60	1.44	17.9					
Spi1-5	M75717		1.65			8.44					
Spi1-6	X00945					67.2					
Tacstd2	AI563854		18.1								
Thra	AI834950	3.38	0.79	0.93	0.96	1.61	1.30	1.20	0.52	1.43	1.84
Tle1	U61362	0.908	3.76	0.69	0.95	1.13		1.74	1.33	1.15	0.68
Tle4	U61363		3.35	0.87	1.12	0.94		0.94		0.97	
Tm7sf	AI060729		1.42	2.10	3.70	3.11		1.00	1.48	1.38	
Tollip-pending	AI842752		2.31								
Tyrobp	AF02463	0.69	3.35	4.20	4.57	2.95	0.77	3.69	2.96	1.89	
Ulk1	AI850194	1.70	3.04	0.51	1.17	0.87	1.34	1.52	0.70	1.12	0.77
	AF053756	2.47	2.80				1.93			0.93	
Wisp1	AF10077			2.01	8.70	4.15			2.16	2.47	
Wrch1-pending	AV246963		2.21								
Transcription											
Alrp	AF041847	0.90	36.3	16.6	12.8	2.45	4.05	8.93	19.6	3.83	0.62
AML1	D26532		2.79	3.79	3.72	3.31			1.86		
Atf3	U19118	6.87	11.9	1.90	4.58	1.81	7.31	3.16	4.93	3.89	1.28
	U19118	17.7	18.1		9.45		13.5	5	7.35	6.43	

Table 2 (cont'd.)

Cebpd	X61800	3.84	13.5	1.36	1.25	0.97	7.03	4.35	1.71	1.01	0.61
c-myc	L00039	2.61	6.71	3.09	2.20	0.88	2.67	2.38	3.91	1.64	
Cnot7	AI931748	2.62		1.45	1.65			2.06	2.42	1.35	
Dlxin-1	AB02944		0.80	1.58	3.34	2.43		1.59	1.36	1.62	
Egr-2	M24377		1.77	1.94	2.84	2.27			2.04	2.30	
Eif1a	AI132207	1.56	4.75	1.65	1.23	1.46	1.06	2.12	2.33	1.34	
	AF026481	0.733	3.46	1.43	1.34	0.84	0.92	1.63	1.52	1.14	0.29
Eif4ebp2	U75530	2.20		2.13	1.30	1.27	2.26			1.57	
	AI848377	31.5				22.1					
Elk1	X87257	3.29		1.25	1.56	1.47		1.82			
En1	L12703	2.5			3.29						
Ets2	J04103	0.98	3.05	1.37	0.86	0.91	1.43	1.52	1.38	1.15	0.79
Fnbp2	L29454		6.0	8.47	11.8	8.85		9.74	7.88	8.11	
Foxl1	X92498	2.06						1.02	0.92		2.08
Fos	V00727	17.9	12.8	8.15	6.38	3.29	5.68	10.5	13.1	16.1	2.51
Fosl1	AF01712		25.1					1.61			
H3f3b	X13605	2.84	4.95	2.68	4.16	3.50	1.41	3.54	3.48	3.01	1.05

Table 2 (cont'd.)

Hey1	AW214298			3.70	4.69	2.98			2.00	2.02		
Hmx3	X75330				3.38							
Ier3	X67644	2.18	4.88	1.94	1.02	0.71	2.57	2.51	1.47	1.03		
Junb	U20735	139	74.7	44.7	27.4		140	31.4	24.3			
		7.47	5.17	2.31			7.00					
Klf3	U36340		5.92		8.15	8.47						
Krox-24	M28845	8.46	5.33	2.96	3.45	1.71	5.02	3.55	2.56	4.51	1.44	
Ler2	M59821	3.06	2.70	1.62	1.46	0.96	1.47	1.55	1.71	1.74	0.70	
Mail-pending	AA614971	3.26	3.61	2.17	1.62	1.60	1.61	2.62	2.75	1.82	0.80	
Mef2a	U94423	2.07	0.72	1.41	1.1	1.4				1.69	1.93	
Mpg-1	L20315	0.93	1.79	3.72	6.06	3.46		2.18	2.44	2.01		
Mth1	AV349001			4.92								
Myf5	X56182		1.06	1.51	3.58	1.60		1.38	0.89	1.69		
Myf6	X59060	0.93	7.02	3.11	1.46	1.0	2.52	2.36	3.11	1.59	0.58	
Myod1	M18779	1.08	2.75	2.29	2.27	1.52	1.76	1.80	1.27	1.50	1.68	
Myog	X15784			3.69	2.97	2.72			3.98			
Ncoa1	U64828	2.63	0.99	1.10	1.32	1.61		1.37	1.32	1.5		
Nfatc2	U36575	3.02			1.92	2.68	4.22		2.81	1.98		
Nfil3	U83148	1.86	6.64	0.97	1.15	0.72	2.26	2.21	1.68	0.79	0.99	
Nr4a1	X16995	2.40	0.56	0.38	0.75	0.66	1.11	0.52	0.64	1.45	0.78	
OxyR	L35599	3.99	4.51	1.44	1.03	1.64	3.21	1.68			2.94	
Peg3 (Zfp)	AF03893	1.42	1.19	1.25	7.66	5.42		1.42	1.73	1.99		
	AW12087	0.51	1.38	1.39	6.47	3.40		1.12	2.29	2.38	0.64	
Pole3	AA83946	2.64	1.03	1.59	1.55	0.97	0.85		0.97	1.24		
Rnf4	AV37235	12.08										
Rrm2	M14223		2.58	6.06	2.95	1.67		2.81	2.39	1.33		

Table 2 (cont'd.)

Sap30-pending	AF075136	1.39	2.36	1.74	2.71	1.97	1.13	2.58	1.98	1.02	1.47
Sox4	AW12415	1.50	1.55	0.90	2.08	2.60	3.42	0.88	0.86	1.40	0.85
Sox11	AF009414			1.94	14.1	3.56			2.27	2.18	
Zac1	X9504			4.26	77.8	58.7				14.4	
Zep	AB013357	0.397	2.03	0.85	0.98	0.75	0.52	0.95	0.85	1.09	0.43
Zfp36	X15378	6.36	3.75	2.79	1.73	2.04	3.28	2.72	1.67	2.31	
Other functions and ESTs											

Table 2 (cont'd.)

Acinus-pending	AI839299		1.24	2.14	2.32	1.78		1.69	1.14	1.19	
Alb1	X13060		6.79			33.8					
ADFP	M93275	1.43	3.29	2.29	0.95	1.36		1.19	1.60	1.29	1.08
Anp32	U73478		2.15							2.695	
Arl6ip	AW122878		1.04	3.34	1.07	1.15		0.84	1.61	1.03	
Calm4	AI119347		2.37		2.51						
Chi3l3	M94584		19.6	11.4				9.97	4.94		
Clca3	AV373378		2.03								
Cldn5	U82758	2.18	2.68	1.81	1.62	2.18	2.56	1.40	1.74	1.86	
Cors-pending	AI315647	4.88	2.16	6.50	177	75.2			7.15	36.5	
Debt	AI841137				4.04						
Dlk1	Z12171	1.04	0.94	0.84	1.99	2.76	1.20		0.71	1.27	1.24
Dscr1	AI846152	1.46	4.95	0.78	0.68	0.47	2.16	1.90	0.93	0.99	0.82
F2	X52308					26.8					
Fga	AI876446		7.45			11.6					
Frg1	U62105		2.03	2.49	1.73	1.90				2.66	
Fxr1h	AV368725	2.93	1.89	1.34	1.63	1.49		2.58		1.65	3.94
Fxyd5	U72680		2.20	2.51	2.39	2.14		1.76	1.68	1.01	
Gbas	AJ001261	2.08	2.40	0.85	1.32	1.58	3.05	0.82	0.77	2.02	2.19
Gc	M55413					32.0					
Gltp-pending	AI842825	0.72	1.56	2.33	2.78	2.45		0.67	1.18	1.28	0.70
Krtdap	AA726579		17.7								
Fgb	AI196896					22.1					
Flg	J03458				19.0						

Table 2 (cont'd.)

Hpxn	U89889					54.8					
	AV105397		1.62			3.77					
Kcnn4	AF042487			2.37		1.89					
Krt1-10	V00830	1.14	10.2	0.81	8.04	1.41		1.77	12.3	1.4	
Lag	AI838080			2.44	2.32						
Lrrn1	AV221593				46.6						
Maged2	AI851574	0.99	0.70	1.35	6.28	4.09	1.14	0.83	1.06	2.64	1.30
mafK	D42124	1.911	3.06				2.152				
Madh1 (mSmad1)	U58992	1.40	4.36	2.25	2.97	1.62	1.87	1.92	1.57	2.05	1.63
MAM	M93264		7.19			29.7					
Meg3	Y13832				52.1	51.5					
Midn-pending	AW124785	1.72	2.37	1.00	1.24	1.0	3.15	0.96	1.06	1.01	1.24
Mpg-1	L20315	0.93	1.79	3.72	6.06	3.46		2.18	2.44	2.01	
mPHLL2	AB003433	4.13	2.18	2.13	1.64	1.58	3.69		1.64	1.34	3.75
MsytIII	D45858	2.42	1.14		1.14	1.42		1.20			
Mup1	AV355798	1.20	0.56	0.38	0.47	2.56	0.57	0.53	1.2	1.90	0.87
	M16359	0.92	1.03			4.25			1.01	2.08	
Mup3	M16357	1.34	0.61			4.41		0.34	1.07	1.48	0.82
Mup5	M16360	1.54	0.61		0.41	3.20			0.66	1.60	0.77
NG22	AF109906	1.8	5.39	0.89	1.3	0.78	0.72	0.99	1.00	0.92	0.73
Npm3	U64450			3.82	4.78		7.76				
Npn3	Z31362	1.27	19.4	2.02	1.09	1.32	4.18	5.07	4.05	1.80	1.85
Obp1b	Y10972	11.46									
Orc2	AV094683		25.1								
Pancortin	D78265	9.51	4.31	8.90	6.86			8.87			
Peg1 (mest)	AF017994		1.06	1.56	15.5	6.5		0.67	0.74	2.30	

Table 2 (cont'd.)

Raet1c	D64162		1.90	2.22	1.07			2.11			
Rnu22	AA684508	0.74	3.44	1.34	1.28	0.94	0.98	1.40	1.57	1.22	0.61
Sid1334	AB025409			2.72	2.32						
shrm	AI641895		2.15	1.46	1.66	1.07		0.81	2.21	1.07	
Slc20a1	M73696		2.18	0.93	1.28				1.14	0.88	
Sp100	AF040242			4.74							
Sprr1a	AF057156		16.3								
Sytip-pending	X52102		2.25	0.86	0.79					0.76	
Tc10l-pending	AW121127	4.80	3.90	2.87	2.63	3.54	6.76	2.75	1.39	1.79	3.11
Tm4sf7	AW124470	2.35	1.67	1.21	0.71		1.06	0.99	1.28	0.89	
Trt	AW122985	1.15	1.44	2.25	1.65	2.03		1.17	1.09	0.92	
Ubc (Ubiquitin C)	AV305832	1.90	4.35	0.78	0.77	0.86	1.80	1.51	1.08	1.30	1.28
Xin	AF051945	1.81	4.12	0.84	1.29	0.8	3.54	1.82	1.23	1.09	0.88
	AA002843	4.73	1.05	1.19	1.97	3.71	2.85	0.76	0.67	0.93	1.81
	AA068153			3.45							

Table 2 (cont'd.)

	AA104818		3.39			3.36					
	AA184423			11.2	14.6			19.4			
	AA189811		1.17	2.41	2.79	2.41				1.17	
	AA407599		2.61	2.27		1.84		3		1.85	
	AA726837		12.7						7.95		
	AA856349			2.85	2.71						
	AI047331					2.22					
	AI447305		4.07	5.91	3.56			5.28	3.09		
	AI450597			5.10	4.43	3.57		3.86	4.71		
	AI451008				7.98						
	AI152789	0.56	2.22	1.21	1.17	1.02	1.14	1.03	0.63	0.57	
	AI834849			5.19	6.53	6.70					
	AV212851				3.63						
	AW04723		1.23	4.02	5.29	2.08		1.14	3.49	2.04	
			0.94	1.93	2.58	1.50		0.77	1.46	0.98	1.43
	AW060457			3.53	4.55	3.28		2.44	2.07	1.8	
	AW107922				4.73						
	AW215456			2.33	3.19	2.90			2.94		
	C85523	4.86	11.3	4.0	1.62	1.45	5.8	5.48	3.84	1.29	0.91
	X58196	1.05	0.68	1.22	4.15	2.64	0.75	0.88	0.97	2.29	0.96
AA407323	AA866768				6.54						
AA407887	AA600542		138		56.9			42.6	80.0		
AA407948	AI836610		0.87	2.02	4.04	2.57	3.64			1.25	3.58
AA409629	AA409629			5.404							
AA409661	AA718792		2.10								
AA410133	AA66710	14.1		5.47	6.01	9.77	6.09		8.7	8.53	24.7

Table 2 (cont'd.)

AA410048	AW259499				3.07	1.77			3.11			
AA536743	AA623587	1.10	2.31	1.06	1.4	0.99	1.31	0.93	1.17	1.00	0.59	
AI035637	AI842259		1.53	3.67	4.18	3.28		1.19	2.69	2.18		
AI132321	AW123773			2.20	3.22	2.00		0.84	1.85	1.49	0.79	
AI323667	AI323667		10.8			3.80		4.07				
AI596360	AV376312	1.01	4.26	0.69	1.31	0.92	1.44	1.39	1.24	1.19	0.60	
AI173274	AI642389		1.17	1.69	3.56	2.91		1.06	1.55	2.38	0.96	
AI413331	AA796989	1.15	0.69	1.38	3.34	3.60	0.92	0.78	1.16	2.69	1.38	
AI482343	AW123850	0.79	0.96	1.17	3.44	2.37	0.93	1.27	1.08	1.36	0.71	
AI585872	AI585872		2.15									
AI596360	AI596360	1.05	5.37	1.18	1.53	0.68	1.93	2.14	1.39	1.28		
AI597080	AI606103				2.82							
AU016206	AI841579		3.92									
AU016588	AI593640		0.49	1.22	2.68	2.78		1.13	1.62			
AU021460	AI131895		2.13	2.01	1.75	1.32		0.91	1.46	1.03		
AU022349	AW046442	2.52	1.45	1.09	0.83		1.03	1.59	1.61	1.27	0.76	
AU044290	AI843106	0.89	3.71	1.02	1.15	1.28	1.56	1.50	1.52	1.17	0.68	
AU046135	AI842065	0.44	0.95	1.67	3.01	2.03	0.19	1.11	2.16	1.68	0.31	
AW122239	AW122239		1.56	3.3	2.73	1.57			3.14	1.2		
AW558171	AW12086	3.4	5.14	1.40	1.46	0.86	3.30	1.58	1.57	0.79		
BB165529	AA275196				2.90				1.76			
C76919	AV349170		1.73	3.43								
C78013	AW124082				11.5	14.0						
C79529	C79529	2.24	1.37	1.28	1.62	1.71		1.14	1.19	1.63		
C79684	AW047929		2.86		3.59				3.99	4.07		
CD84	AA815831		3.92	3.67	4.13							

Table 2 (cont'd.)

D1Ucla3	AI182073					5.901						
D4Ertd117e	C77296		7.86							5.83		
D7Ertd183e	C78535		4.75			3.41						
D8Ertd69e	AA543502	2.11	0.45	1.64	0.80			1.05	0.78	1.68		
D15Ertd781e	AI528219	2.87	1.39	1.63	2.20	2.01	1.83	1.42		1.2		
M32486	M32486		12.2	7.31	14.2	9.14		5.35	11.5	8.83		
R7539	AI852838	1.13		0.86	5.88	5.74		1.00	0.86	2.54	1.25	
0610011I04Rik	AI787183		1.29	3.06	2.04	2.38		1.54	3.45	1.44		
0610012A05Rik	AA815845	1.15	3.16	0.82	0.72	1.02	1.25	1.48	0.69	0.88	1.24	
1110007F23Rik	AV366654				3.39	3.01						
1110008G13Rik	AI838513				25.9							
1110032C13Rik	AI847051		2.11	0.88	1.07							
1110038L14Rik	AA681998			4.98	3.53	2.39			3.94			
1110064N10Rik	AW124599	0.79	2.83	1.29	1.55	0.81	1.34	1.30	2.16	1.87	0.83	
1190002H23Rik	AI854358	1.48	6.3	1.54	0.96	0.47	1.46	3.10	1.05	1.08	0.58	
1500011E11Rik	AI848915	5.23	2.76	3.77								
1500031M19Rik	AV230529			9.24								
1600012H06Rik	AW011716		2.11	1.97	3.0	2.50		1.26	2.62	3.07		
1600023E10Rik	AI849082	1	3.44	2.67	3.38	2.39	0.63	1.89	2.53	1.43		
1700017B05Rik	AW049360		1.70	1.91	2.81	2.45		1.37	1.51	1.37		
1810003P21Rik	AI844626	0.77	0.41	1.18	3.97	2.74	0.46	0.74	0.91	1.55	0.56	
1810012N18Rik	AI839212	0.66	2.53	0.77	0.85	0.77	0.89	1.07	1.02	0.89	0.45	
1810027D10Rik	AI504305	0.21	1.89	7.38	5.84	1.50		3.62	4.7	0.96	0.32	
1810045K17Rik	AI852409		1.97	2.23	1.03	0.79				1.31		
1810049E02Rik	AA763937	0.91	1.48	1.76	2.03	2.20	1.32	1.58	1.49	1.15	0.79	
2010015J01Rik	AI844812	2.29		1.37		1.15		1.82		1.11		

Table 2 (cont'd.)

2010107K23Rik	AA710297				3.57	7.02				2.50		
2300002G24Rik	AI604345		18.6		7.21				10.7			
2300003P22Rik	AA727291		3.85						2.28			
2310020A21Rik	AI173973		2.38									
2310021G01Rik	AI606257		2.22	1.35	1.71	1.25			2.51	1.05		
2310021M12Rik	AI834895	2.35	1.19	1.51	1.56	1.81		1.34	1.08	1.60	1.17	
2310038G18Rik	AI851313	2.48	1.15	2.11	2.09	1.09		1.51	0.91	1.63		
2310046B19Rik	AA612450	0.96	0.75	1.10	1.84	2.21	0.42	1.14	0.92	1.95	0.76	
2410001E19Rik	AV354124			1.67	2.16	4.05		4.56		3.22		
2410018G20Rik	AI852414		2.68	0.85	0.66		1.47	1.06	0.93	1.05		
2410045D21Rik	AW047616	4.01	1.66	2.98	3.39	2.25						
2610024P12Rik	AW124113	1.20	3.88	4.47	4.34	4.34		2.39	2.36	1.90	0.94	
2810052M02Rik	AW061307				3.40	3.89						
2810052M02Rik	AI852196				2.59							
3000002J10Rik	AI844911		0.61	1.02	2.53	1.42	0.61	0.86	0.64	1.17	1.13	
3010002H13Rik	AA986050					33.8						
3110023F10Rik	AA867778	1.88	0.74	2.14	2.61	3.42	1.41					
3110038L01Rik	AW124483	2.21	3.78	1.51			4.13	1.49		1.06		
	AI844396	0.89	3.42	1.27	0.81	0.40	1.30	1.46	1.27	0.97	0.46	
3230401N03Rik	AI747444	0.57	0.71	1.42	2.65	1.35		0.73	0.85	1.13	0.96	
3230402E02Rik	AA623874	1.13	3.94	1.74	1.58	1.37		1.84	1.74	1.32		
3300002C04Rik	AA874490		2.80	2.26			1.82		2.01			
4432405K22Rik	AI037493	0.76	3.54	0.98	0.96	0.65	0.86	1.31	0.75	1.23	0.42	
	AI194333	0.85	3.64	1.15	0.96	0.83	0.91	1.34	1.22	1.20	0.43	
4632415A01Rik	AW124460	2.26	1.02	1.06	0.84	1.37				1.04		
4833432B22Rik	AW046889			3.23	3.15					4.46		

Table 2 (cont'd.)

4921531N22Rik	AI196645		1.10	1.43	2.20	2.01		1.39	1.33	1.66	
4930534K13Rik	AW125713		2.14	1.76	1.54	1.23			1.03	0.72	
4933428G09Rik	AI595812	1.71	1.65	1.54	2.58	1.58		1.41	1.38	0.89	
5430432M24Rik	AI845815				2.86	1.98					
5730437E04Rik	AW046857	2.71			3.93	2.00				1.23	
5830413E08Rik	AI849939	4.73	4.26	0.99	1.14	1.01	3.91	1.95	1.47	0.74	0.91
6530405F15Rik	AI644072	1.29	0.77	1.52	2.89	2.90		1.32	1.53	2.41	
8430417G17Rik	AI225296	3.43	7.19	1.18	0.82		4.68	2.77	1.98	1.03	

Table 3. Down-regulated genes following femoral artery ligation

Gene Name	Accession Number	Femoral artery ligation					Sham				
		6 hour	1 day	3 day	7 day	14 day	6 hour	1 day	3 day	7 day	14 day
Cell growth and survival											
Cdc2l1	M58633	0.40	1.25	0.63	0.74	0.73		0.84	0.92	0.90	
Gdap1	Y17850	0.66			0.49	0.38	0.77		0.47	1.10	0.88
Map2k3	AI852636	1.14	1.30	0.63	0.47	0.67	1.45	1.06	0.67	0.96	0.88
Map2k6	X97052	1.04	0.68	0.36	0.40	0.71	1.12		0.45	1.59	0.72
Pkia	M63554	0.96	0.62	0.28	0.42	0.97	0.86	0.82	0.41	1.07	0.78
Orc5	AJ007360	0.35	0.78	0.59	0.33	0.44		0.71	0.67	0.92	0.47
Cell shape and motility											
Acta2	X13297	0.52	0.29	0.41	1.00	0.55	0.34	0.87	0.57	1.40	0.52
Actb	M12481	0.80	1.82	0.37	1.41	1.17	1.40	0.69	1.11	0.99	0.80
Ank1	U76758	0.74	0.26	0.33	0.53	0.68	0.98	0.53	0.46	1.32	0.73
D4Mil1e	D17577	1.32	0.32	0.59	0.52	0.92	0.91	0.85	0.54	0.98	1.04
MLC1s, M	X12972	1.17	0.80	0.08	0.04	0.21	0.78	0.99	0.19	0.41	0.59
Myhcb	AJ223362	0.67	0.73	0.11	0.11	0.19	0.63	1.46	0.30	0.61	0.42
Myh11	D85923	0.61	0.33	0.25	0.27	0.30		0.75	0.42	1.17	0.62
MYOC	AF041335	1.06	0.37	0.37	0.58	0.71	1.13	0.77	0.59	1.27	0.80
myosin l	M91602	0.62	0.55	0.04	0.07	0.12	0.50	0.94	0.12	0.46	0.29

Table 3 (cont'd.)

Plec1	AW123286	0.50	1.23	0.77	1.26	0.50	1.34	1.39	0.53	1.34	0.72
Pfn2	AW122536	0.63	0.44	0.52	0.51	0.48	0.61	0.89	0.63	1.32	0.63
Tagln	Z68618	0.88	0.43	0.64	0.63	0.69	0.70	0.90	0.58	1.36	0.94
Tpm5	U04541	0.64	0.74	0.14	0.14	0.18	0.55	1.14	0.32	0.55	0.35
Cytokines and inflammation											
Adn	X04673	0.55	0.59	0.54	0.40	0.63	0.31	0.56	1.22	0.87	0.69
FAT (CD36)	L23108	0.54	0.77	0.75	0.26	0.51	0.50	0.84	1.11	0.93	
Igh-6	V00821	0.54	0.52	0.32	0.17	0.43	0.61	0.27	0.73	0.80	0.76
	V00817	0.72	0.40	0.46	0.21	0.53	0.70	0.27	0.82	0.70	0.86
Igk-V28	M18237	0.02	0.02	0.50	0.02	0.04			1.05	0.04	
J chain	M90766	0.25		0.23	0.17	0.26	0.37	0.19	0.59	0.16	
Ly6	X04653	0.49	2.33	2.04	0.69	1.00	1.02	1.91	1.77	1.25	0.71
Scya11	U77462	0.78	0.82	0.46	0.39	0.48	0.44	0.70	0.70	0.97	0.53
SLC	AF035684			0.73	0.31	0.74		0.54	0.86	0.83	
Extracellular matrix											
Col6a3	AF064749	0.79	0.42	1.4	3.07	3.25	0.51	0.90	1.16	1.86	0.74
Gzmg	J02872			0.79	0.61	0.46	0.87			0.70	
Matn2	U69262			0.40	1.51	1.41		0.47	0.81	1.01	0.50
Ogn	AA647799	0.63	0.24	0.61	2.35	1.65	0.28	0.80	0.86	2.14	0.57
Og	D31951	0.44	0.29	0.55	1.74	1.82	0.59	0.71	0.73	1.74	0.52
TIMP-3	U26437	0.47	0.47	0.35	0.40	0.48	0.58	0.60	0.58	0.92	0.35
Metabolism											

Table 3 (cont'd.)

ab, SCD, Scd-1	M21285	0.61	0.48	0.62	0.41	0.54	0.38	0.43	0.64	1.01	0.63
Adsl	AA606587	0.38	0.63	0.39	0.47	0.40	0.44	0.71	0.66	1.16	0.41
Ahd-2	M74570	0.53	0.72	0.48	0.37	0.60	0.48	0.77	0.85	1.05	0.53
Amd-1, AdoMetDC	D12780	0.87	0.54	0.19	0.28	0.41	0.94	0.66	0.40	1.13	0.76
Amd2	Z23077	1.32	0.43	0.15	0.23	0.44	1.06	0.55	0.23	0.93	0.78
	Z23077	1.86	0.42	0.13	0.25	0.44	1.49	0.44	0.16	0.83	0.82
Amy1	J00356	0.47	0.96	0.47	0.53	0.74	0.67	0.78	1.05	1.38	0.75
Aoc3	AF078705	0.44	0.23	1.0	0.61	0.66	0.52	0.67	1.49	1.15	0.42
Ap2, Lbp, ALBP/Ap2	M20497	0.74	0.64	0.35	0.61	0.61	0.59	0.57	0.85	0.88	0.57
Apobec2	AW124988	0.61	0.57	0.20	0.46	0.40	0.52	0.51	0.48	0.77	0.40
Cas1	M29394	0.44	0.76	0.48	0.52	0.59	0.24	0.85	1.12	0.63	0.35
CD26	U12620				0.31	0.55				0.77	
Ces3	AW226939	0.25	0.21	0.26	0.19	0.20	0.19	0.34	0.75	0.91	0.36
	AW226939				0.16				0.85	0.75	0.54
Cdo1	AI854020	0.50	0.19	0.42	0.33	0.43	0.17	0.47	0.99	0.80	0.51
Ckmt2	AV250974	0.55	0.50	0.13	0.22	0.28	0.46	0.54	0.22	0.62	0.45
Cyp2e1	X01026	0.30	0.14	0.25	0.18	0.32	0.27	0.32	1.44	0.85	0.37
Cox8b	AV260484	0.68	0.91	0.40	0.50	0.44	0.91	1.04	0.50	1.19	0.69
	U15541	0.62	0.76	0.33	0.46	0.35	0.84	1.13	0.54	1.05	0.68
Cpa3	J05118		0.66	0.64	0.35	0.74		0.68	2.65	1.39	
Cycs	X01756	0.53	0.59	0.22	0.42	0.36	0.47	0.49	0.46	0.93	0.42
Dia4	U12961		0.78	0.76	0.28	0.34		1.27	1.22	0.80	0.68
	U12961	0.82	0.93	0.65	0.34	0.52	0.58	1.08	1.26	0.90	0.73

Table 3 (cont'd.)

Ephx2	Z37107	0.72	0.57	0.41	0.32	0.35	0.46	0.71	0.54	0.78	0.53
Gcdh	U18992	0.84	0.90	0.40	0.73	0.83	0.76	0.88	0.91	1.18	0.66
Gdm1	D50430	0.74	0.31	0.45	0.44	0.47	0.59	0.81	0.57	1.08	0.49
Enpp2	AW122933	0.49	0.39	0.83	0.31	0.55	0.42	0.53	2.01	0.69	0.38
Fasn	X13135	0.65	0.14	0.38	0.26	0.33	0.25	0.25	0.54	0.58	0.70
Fbp1, Fb	D42083	0.61	0.66	0.25	0.28	0.36	0.56	0.51	0.45	0.69	0.59
Fmo1	D16215	0.32	0.19	0.41	0.50	0.41	0.16	0.62	0.83	0.82	0.31
Gdc1	M25558	0.68	0.49	0.24	0.34	0.68	0.87	0.43	0.23	0.99	0.74
Gdm1	D50430	0.74	0.31	0.45	0.44	0.47	0.59	0.81	0.57	1.08	0.49
Glut4	M23383	0.98	0.54	0.43	0.35	0.46	1.11	0.70	0.48	0.85	0.90
Hadh	D29639	0.73	0.53	0.39	0.45	0.62	0.59	0.87	0.41	0.99	0.61
Hmgcl	U49878	0.48	2.30	1.00	0.72	0.64	1.50	1.29	1.84	1.64	0.79
Hsd11b1	X83202	0.71	1.15	0.80	0.39	0.66	0.58	1.00	1.81	0.93	0.69
Hsd17b4	X89998	0.40	0.75	0.63	0.40	0.45	0.46	0.70	0.85	0.82	0.51
Ldh2	X51905	0.82	0.58	0.14	0.21	0.30	0.61	0.56	0.29	0.60	0.41
Lnap1	AF023463	0.61	0.71	0.46	0.36	0.55	0.54	0.62	0.56	0.80	0.51
Lpl	AA726364	0.56	0.65	0.41	0.42	0.42	0.52	1.00	0.85	0.69	0.49
Mccc1	AW123316	0.72	0.52	0.57	0.62	0.48		0.84	1.08	1.32	0.97
Mod1	J02652	0.52	0.68	0.44	0.34	0.57	0.55	0.64	0.74	1.08	0.97
Pck1	AF009605	0.58	0.34	0.43	0.41	0.45		0.81	1.13	0.58	0.58
Pgam2	AF029843	0.72	0.45	0.32	0.40	0.79	0.78	0.51	0.34	1.12	0.77
Phkg	J03293	0.72	0.56	0.29	0.43	0.56	0.77	0.48	0.46	0.87	0.69
Phka1	X74616	1.03	0.75	0.41	0.48	0.77	1.18	0.80	0.42	1.18	0.83

Table 3 (cont'd.)

Ppara	X57638	1.07	0.70	0.38	0.37	0.44	1.24	0.62		0.77	0.66
pdha-1	M76727	0.68	0.88	0.34	0.43	0.48	0.70	0.62	0.53	0.95	0.61
Psmb4	AA638816	1.17	0.47		0.48				0.56	0.73	
Siat10	AI153959	0.87	0.69	0.43	0.43	0.67	0.90	0.68	0.49	1.63	1.06
Sucla2	AF058955	0.79	0.64	0.31	0.64	0.54	0.81	0.87	0.65	0.69	0.71
Temt	M88694	0.38	0.34	0.42	0.43	0.37	0.36	0.92	0.85	0.87	0.50
Timm10	AW122428		0.55	0.42	0.83	0.64		0.90	0.88	1.02	
Tpi	L31777	0.69	0.65	0.28	0.46	0.84	0.76	0.67	0.38	1.06	0.86
Ucp	AV294354	0.57	0.46		0.45	0.60		1.16			
Signaling											
CD106, VCAM-1	M84487		0.31	2.2	2.76	1.30		0.81	3.91	1.08	
Epcr	L39017	0.90	1.28	1.28	0.48	0.85	0.64	1.06	1.42	1.42	0.81
Gnai1	AI153412		0.37	0.61	0.41	0.38		0.38	0.89	0.89	
IGFBP-5	L12447	1.06	0.29	1.5	1.39	1.22	1.17	0.79	1.24	1.68	1.54
Irf1	M21065	1.03	0.65	1.11	0.73	0.31	0.79	0.66	0.73	0.90	
Fzd9	Y17709	0.88	0.61	0.59	0.50	0.76	0.87	0.57	0.70	1.08	1.33
Mlf1	AF100171	0.91	1.22	0.36	0.27	0.51	0.89	1.02	0.48	0.97	0.68
Nore1-pending	AF053959	0.40	1.08	1.11	0.94	0.73	0.82	0.72	1.59	0.68	
pgk1	M15668	0.82	0.76	0.39	0.38	0.65	0.60	0.85	0.53	0.96	0.65
Pparg	U10374		0.23	0.61	0.46	0.38		0.74	0.51	0.51	
Ptb2-pending	AI119718		1.26	0.45	0.64	0.84		0.82	1.20	1.19	
PTHRP	M60057	0.63	0.41	0.36	0.73	0.78	0.54	0.67	0.79	0.76	0.80
Rasd1	AF009246	0.74	0.26	0.34	0.13		0.35		0.45	0.47	

Table 3 (cont'd.)

S100a1	AF087687	0.52	0.77	0.49	0.48	0.45	0.84	0.81	0.70	0.97	0.52
Slc25a15	AA986782	0.46	0.45	0.59	0.48	0.67	0.61	0.48	0.43	1.24	0.64
Slc25a11	AW049350	0.73	0.49	0.31	0.40	0.55	0.78	0.56	0.37	0.92	0.67
Styx	U34973	0.67	0.31	0.43	0.51	0.36	0.55	0.69	0.65	1.23	0.78
Thrsp	X95279	0.50	0.18	0.35	0.30	0.30	0.29	0.25	0.51	0.59	0.62
Transcription											
Ankrd2	AJ011118	0.85	2.75	0.86	0.17	0.23	1.33	2.38	0.79	0.39	0.44
C1d-pending	X95591	0.37	0.58	0.41	0.85	0.59	0.46	0.56	0.88	0.77	0.46
H2afy	AA646966	0.38	0.65	0.34	0.84	0.46	0.22	0.24	0.80	0.81	0.31
Hist4	M32459	1.29	0.87		0.51	0.39	1.01	0.93	0.26	0.81	0.99
Hoxa10	L08757		0.50	0.54	0.30	0.74	0.61		0.58	0.88	
Hoxd8	X56561	0.58	0.56	0.36	0.29	0.93	0.59	0.38	0.39	0.67	0.58
Meox2	Z16406		0.39	0.78	1.00	0.80	0.10	0.86	0.53	0.91	0.21
Satb1	U05252	0.83	0.59	0.33	0.45	0.39	0.42	0.57	0.66	0.94	0.53
Sox18	L35032	0.19	0.62	0.93	0.57	0.78	0.64	0.52	0.60	0.71	0.64
Spnr	AI838709		0.55	0.59	0.41	0.45	0.71	0.82	0.84	1.00	0.54
Crtr1-pending	AA734817	0.82	1.55	0.49	0.43	0.66	2.39	0.86	0.87	0.98	0.99
Other functions and ESTs											
adrenodotoxin	L29123	0.49	0.48	0.38	0.35	0.40	0.47	0.65	0.52	0.81	0.42
Akl3l-pending	AI854743	0.53	0.34	0.55	0.42	0.41	0.39	0.48	0.65	1.10	0.34
Ank	AW049351	0.64	0.62	0.41	0.37	0.35	0.86	0.74	0.52	0.95	0.53
Aqp4	U48398	1.03	0.23		0.13	0.39	0.99	0.15	0.20	0.66	0.35

Table 3 (cont'd.)

Aqp4	U88623	0.77	0.09	0.13	0.12	0.39	0.59	0.10	0.17	0.57	0.43
AQ1	L02914	0.99	0.97	0.82	0.49	0.59	1.71	1.14	0.83	1.06	1.07
AREC3	D50418	0.48	0.39	0.61	0.82	0.56	0.51	1.02	0.74	1.02	0.68
Blcap	AW121500	0.72	0.83	0.57	0.51	0.32	0.99	0.49	0.66	0.89	0.74
Bnip3	AF041054	0.56	1.53	0.42	0.41	0.49	0.73	0.88	0.58	0.84	0.53
Brd7	AW125534		0.82	0.35	0.65	0.78	1.19	0.76	0.78	1.04	
Cd24a	M58661	0.59	0.88	0.92	0.29	0.34	0.57	1.09	1.22	0.65	0.57
D11Bwg13	AW121381	0.68	0.79	0.38	0.44	0.52	0.85	0.72	0.46	0.94	0.69
D14Ertd1	AW123154	0.27	1.12	0.56	0.82	0.80	0.86	1.32	0.92	0.85	0.57
EIG 180	AB023957		0.38	0.77	0.47	0.52		0.81	0.57	0.88	0.80
ENDOG	AB012108	1.16	0.73	0.65		0.50		0.63	0.58	0.93	
Etl1	X69942		0.86	0.41	0.90	0.86		0.79	0.91	0.84	
Fem1a	AI836048	0.77	0.71	0.33	0.48	0.56	0.87	0.68	0.41	1.05	0.74
Fsp27	M61737	0.50	0.43	0.68	0.24	0.56		0.70	1.21	0.61	0.43
Mld, shi, hmbpr	M11533	1.13	0.44	0.38	0.27	0.45	0.55	0.38	0.46	0.61	0.78
Mup1	AV355798	1.20	0.56	0.38	0.47	2.56	0.57	0.53	1.20	1.89	0.87
Mup-1, Up-1	M17818	1.23	0.47	0.38	0.39	2.17	0.61	0.49	1.16	1.41	0.98
Mup5	M16360	1.54	0.60		0.41	3.20			0.66	1.59	0.77
Nedd4a	AV365271		0.55	0.48	0.95	0.76		1.48	1.31		0.93
NLRR-1	D45913	0.55	0.45	1.2	2.80	0.99	0.58	0.99	1.46	1.12	0.72
Nudel-pending	AI837311	0.67	0.94	0.44	0.71	0.74	0.80	0.48	1.04	0.90	0.50
ORF13	AI850202	0.72	0.78	0.33	0.39	0.47	0.80	0.83	0.43	0.99	0.70
Pcm1	AF039021	1.09	0.73	0.34	0.73	0.86	0.75	0.78	0.84	0.76	0.75

Table 3 (cont'd.)

Pgy2	J03398	0.76	0.68	0.27	0.28	0.33	0.74	0.63	0.27	0.86	0.69
Retn	AA718169	0.40	0.40	0.75	0.19	0.44		0.33	1.26	0.78	0.47
S3-12-pending	AF064748	0.75	1.59	0.57	0.26	0.52	1.50	0.70	0.71	0.69	0.58
Sepr	AI840996	0.93	0.95	0.42	0.46	0.64	1.01	0.82	0.44	0.95	0.78
Skd3	AI837887	0.77	1.17	0.58	0.47	0.74	1.07	0.73	0.64	1.63	0.87
Sprr2a	AJ005559	1.87	0.42	1.3	0.88	1.15	1.52	1.36	0.79		1.08
Sui1-rs1	Z50159	0.95	1.10	0.83	0.48	0.55	0.92	1.33	0.94	1.12	0.76
ten-m3	AB025412		0.77	0.82	0.42	1.01			0.76	1.22	
TGN38, TGN38A	D50031	1.84	1.46	1.29	0.48	0.71	1.21	1.05	1.58	1.27	0.97
Trfr	X57349	1.15	0.23	0.38	0.79	0.98	1.39	0.47	0.24	0.78	0.97
Ubce4	X926641	0.24	0.55		0.75	0.35	1.17	0.73	0.87	0.53	1.67
UCP-3	AB010742	1.56	1.96	0.59	0.46	0.74	3.26	0.42	0.72	1.03	0.77
Vdac3	U30839	0.71	.80	0.38	0.45	0.63	0.66	0.80	0.49	1.04	0.59
	AA162144	0.42	0.35	0.40	0.61	0.77		0.87	0.63	0.96	0.49
	AA177382	0.65	1.57	0.51	1.68	0.48	0.74	1.28	1.23	1.12	1.06
	AA666464	0.32	1.07	0.92	1.53	1.33	0.83	1.06	1.69	1.61	
	AI037032	0.59	0.38	0.56	0.29	0.44	0.67	0.76	0.69	0.95	
	AI118905	0.39	0.29	0.32	0.24	0.39	0.40	0.32	0.84	0.50	0.53
	AI194254	0.42	1.05	0.82	1.21	0.49	0.85	0.82	1.15	1.05	0.61
	AI461837		0.78	0.98	0.47	0.51		0.73	0.65	0.99	0.79
	AI504338	0.56	0.81	0.60	0.43	0.37	0.62	1.01	1.08	0.94	0.52
	AI604013	0.43	0.96	0.48	0.64	0.55	0.59	0.98	1.09	1.19	0.64
	AI835081	1.72	0.23	0.87	0.85	1.05	1.83	0.82	0.72	0.45	1.33

Table 3 (cont'd.)

	AI837830	0.87	0.69	0.92	0.69	0.38	0.79	0.70	0.54	0.82	0.91
	AI839175	0.51	0.34	0.70	0.61	0.52	0.17	0.87	0.78	1.07	0.39
	AI839232		0.50	0.48	0.57	0.48		0.90	1.38	0.85	
	AI842938	0.41	0.85	0.75	0.61	0.51	0.87	0.81	1.10	0.85	
	AI846531		0.53	0.67	0.48	0.39			0.48	0.90	
	AI852011		0.89	0.38	0.89	0.92				0.61	
	AI852124	0.46	0.68	0.48	0.47	0.42	0.43	0.83	0.57	0.97	0.43
	AV222871	1.24	0.51	0.71	0.31	0.33	1.47	0.45	0.83	0.64	1.40
	AV319920	1.92	0.40	0.71	0.72	0.64	1.60	1.22	0.53	0.85	0.78
	AV352777		1.24	0.49	1.10	0.79		1.48	1.07	1.08	0.89
	AW047232		0.83		0.37	0.49	0.44	0.73	0.49	0.82	0.35
	AW125043		0.42	0.51	1.40	0.98		0.85	0.75	1.03	
	AW125453	0.31	0.29	0.54	1.96	1.25		0.64	0.61	1.16	0.43
	AW122615	0.65	0.75	0.45	0.41	0.50	0.60	0.85	0.61	0.85	0.53
	X00686		1.73	0.76	0.36	0.55	2.89	1.36	1.02	1.42	1.19
0610006O	AW060827	0.65	0.55	0.39	0.36	0.40	0.65	0.81	0.50	0.89	0.69
0610033L	AI853855	0.65	0.69	0.29	0.49	0.45	0.96	0.78	0.50	1.06	0.82
0610041L	AI839425	0.66	0.73	0.41	0.44	0.47	0.64	0.88	0.54	1.03	0.59
0710008N	AA674669	0.80	0.73	0.36	0.46	0.70	0.83	0.78	0.47	1.06	0.68
1110003O	AI847054	0.56	0.72	1.05	0.49	0.73	0.42	0.83	1.16	1.24	0.57
1110003P	AI835446	0.73	0.56	0.28	0.36	0.54	0.77	0.74	0.26	1.04	0.78
1110004B	AI552528	0.47	0.49			0.87	1.27	1.13	0.60	1.22	0.94
1110004B	AI425990		0.27	0.81	0.67			0.68	0.88	0.80	

Table 3 (cont'd.)

1110007M	AA693236	0.50	0.35	0.45	0.54	0.40	0.45	0.76	0.51	0.73	0.49
1110020E	AI847158	1.39	0.71	0.44	0.50	0.23	0.93	0.58	0.48	0.86	1.13
1110039O	AI845882	0.59	0.86	0.46	0.48	0.37	0.74	1.02	0.85	1.02	0.52
1110049G	AV073962	0.68	0.80	0.44	0.69	0.59	0.79	0.89	0.95	1.04	0.79
1110020A	AW121838	1.17	0.84	0.39	0.41	0.62	1.61	1.20	0.54	0.90	0.62
1110037N	AI852741	0.44	0.50	0.65	0.69	0.70		1.11	0.66	1.06	0.46
1110067D	AW121603	0.93	0.62	0.31	0.36	0.41	0.73	0.99	0.44	1.02	0.60
1200012F	AI844846	0.65	0.62	0.36	0.51	0.46	0.59	0.73	0.75	0.78	0.59
1210001E	AI846595	0.29	1.11	0.57	0.65	0.70	0.78	0.64	0.80	1.00	0.56
1200012G	AA880988	0.80	1.02	2.09	0.47	0.79	0.60	1.45	2.98	1.16	0.60
1300002P	AJ011864		0.70			0.39		1.14	0.76	0.70	
1700016A	AI197431	0.50	0.66	0.88	0.65	0.46	0.55	0.90	0.97	1.05	0.96
1500002K	AW124337	0.48	0.64	0.67	0.39	0.58	0.31	0.73	1.17	0.81	0.45
1810010A	AW122692	0.74	0.76	0.36	0.45	0.57	0.67	0.63	0.38	0.99	0.60
1810015C	AW122893	1.14	3.42	0.83	0.40	0.49	1.62	1.81	0.99	0.84	0.72
1810063B	AW046438	0.74	0.48	0.54	0.61	0.58	0.63	0.99	0.72	0.91	0.53
1810073P	AW124781	1.04	0.76	0.49	0.59	0.39	1.07	0.76	0.76	0.93	1.12
2010200J	AI835436	0.61	0.46	0.49	0.60	0.67	0.42	0.76	1.04	0.99	0.43
2010306B	AI843448	1.10	0.94	0.48	0.49	0.54	1.23	1.00	0.66	1.01	1.00
210420E	AI850195	0.75	1.31	0.37	0.61	0.62		1.02	1.24	0.65	
2310004B	AI845798	0.76	0.88	0.40	0.28	0.28	0.70	0.95	0.42	0.67	0.59
2310004B	AI845798	0.85	1.04	0.74	0.36	0.50	0.99	1.18	0.70	0.80	0.67
2300008A	AI181132	0.89	0.92	0.26	0.32	0.57	0.84	0.93	0.31	1.00	0.74

Table 3 (cont'd.)

2310005P	AI838150	1.01	0.66	0.25	0.47	0.81	0.80	0.90	0.29	1.37	1.03
2310016A	AW049373	0.78	0.63	0.27	0.39	0.52	0.66	0.69	0.39	1.11	0.74
2310032D	AW125284	0.83	0.71	0.35	0.36	0.37	0.29	0.56	0.53	0.77	0.33
2310075M	AW124226	0.79	0.91	0.43	0.49	0.68	0.84	0.88	0.81	1.23	0.71
2410006N	AI853344	0.79	0.90	0.50	0.77	0.88	0.78	0.91	1.07	0.72	
2610001J	AW124115	0.95	1.02	0.82	0.49	0.87	1.03	0.67	0.74	1.02	0.94
2610002K	AI849679		0.93		0.49			0.94		1.06	
2610205H	AW121984	0.79	0.69	0.34	0.49	0.58	0.60	0.84	0.50	1.07	0.50
2610207I	AI648018	0.67	0.77	0.36	0.44	0.63	0.64	0.75	0.40	0.97	0.73
2700023P	AI842066	0.70	0.62	0.38	0.45	0.59	0.74	0.88	0.54	1.04	0.57
2700043I	AI849035	0.37	0.71	0.56	0.92	0.90	0.87	0.87	0.84	1.33	0.57
2810407E	AV299153	0.85	0.44	0.65	0.76	0.68	0.61	1.00	0.74	0.77	0.58
2810422O	AI552570	0.65	0.68	0.72	0.50	0.51		0.91	0.98	1.20	0.73
2810454G	AA874446		1.09	0.37	0.59	0.53		1.33	0.86	0.75	0.87
2810470K	AA867497	0.90	1.21	0.91		0.36			0.97	1.12	
2900024N	AI508500				0.93	0.47			1.20		2.57
2900062L	AI839718	0.20	0.26	0.80	0.27	0.30		0.59	1.38	0.36	0.34
3010033P	AW259500	0.56	0.81	0.58	0.45	0.38	0.82	1.16	0.89	1.03	0.63
4930563P	AW046003	1.58	0.88	0.72	0.38	0.85	1.12	0.68	0.81	0.86	1.37
4930569O	Y08027	0.89	1.04	0.46	0.39	0.53	1.03	0.83	0.66	1.17	0.68
4931430I	AI626942	0.71	0.43	0.39	0.61	1.16	1.06	0.97		1.69	1.33
5730469M	AI850090	0.61	0.35	0.41	0.33	0.26	0.56	0.63	0.68	0.76	0.49
6330416C	AI847486	0.74	0.74	0.40	0.45	0.52	0.77	0.84	0.55	0.98	0.73

Table 3 (cont'd.)

A430101B	AI852768	0.82	0.67	0.78	0.47	0.65	0.92	0.89	1.13	1.00	1.14
AA407980	AA288979	0.68	0.65	0.48	0.50	0.62	0.59	0.75	0.63	1.07	0.62
AA408956	AA408956	0.93	1.69	0.73	0.50	0.39	0.89	1.28	0.73	1.16	0.53
AA409502	AI850948	0.63	0.61	0.34	0.39	0.52	0.49	0.66	0.51	0.88	0.44
AA420417	AW123788	0.25	0.34	0.60	0.73	0.38	0.37	0.75	0.69	1.14	0.28
AA959601	AW125299	0.48	0.69	0.47	0.23	0.36	0.48	0.73	0.80	0.86	0.36
AI115348	AI842192	2.29		0.36	0.49	0.95	0.94	0.64		1.23	1.95
AI225904	AA711773	0.79	0.64	0.83	0.27	0.55	0.77	0.72	1.06	0.66	0.69
AI255373	AW121801	0.82	0.89	0.57	0.38	0.92	0.92	1.37	0.81	1.24	0.49
AI317193	AV367141	0.43	0.73	0.27	0.51	0.51	0.88	0.59	0.88	0.56	0.81
AI327140	AI848393	0.52	1.00	0.73	0.47	0.45	0.81	1.15	0.87	0.96	0.60
AI426782	AA871166	0.56	0.33	0.78	1.00	0.70	0.40	0.75	0.95	0.93	0.51
AI429613	AI606300		0.74	0.83	0.60	0.40	1.32	0.79	0.81	0.75	0.95
AI447096	AI509330	0.57	0.58	0.22	0.42	0.51	0.47	0.51	0.48	0.96	0.52
AI481320	AW046470	0.83	0.67	0.58	0.42	0.56	0.81	0.84	0.64	1.08	0.49
AI551257	AI843063	0.40	0.43	1.1	0.49	0.52	0.57	1.08	0.90	1.01	0.64
AI551766	AW122882	0.57	1.44	0.54	0.50	0.62	0.68	0.85	0.64	1.34	0.63
AI788978	AW125884	0.75	1.61	0.36	0.34	0.38	0.82	1.29	0.60	0.85	0.63
AI848390	AW045204	0.55	0.46	0.23	0.31	0.37	0.66	0.63	0.37	0.83	0.57
AI195826	AW121745	0.41	0.71	0.46	0.77	0.60	0.26	0.68	0.98	1.00	0.23
AI844545	AI844545	0.84	1.19	0.56	0.38	0.58		1.45	0.70	1.05	0.62
AU018239	AW124144	0.87	0.74	0.60	0.47	0.67	0.75	1.14	0.93	1.12	0.58
AU018540	AI848853		0.75	0.44	0.65	0.76		0.68	0.95	0.94	

Table 3 (cont'd.)

AU041772	AW123223	0.59	1.19	0.47	1.03	0.65	0.68	1.07	1.05	1.36	0.76
AV277466	AJ011107				0.38	0.62				1.30	
AW047450	AW047450	0.57	0.40	0.44	0.47	0.49	0.62	0.50	0.51	1.12	0.46
AW061234	AW061234	0.64	0.49	0.52	0.48	0.55	0.63	0.65	0.75	1.09	0.62
AW109744	AA690483	0.52	0.63	0.55	0.36	0.46	0.42	0.79	0.93	0.97	0.40
C80633	AI853240	0.48	0.46	0.57	0.42	0.40	0.40	0.97	0.63	0.90	0.66
N28078	AI835060	0.64	0.53	0.47	0.46	0.54	0.64	0.62	0.62	0.94	0.61